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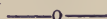
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PART I., 1892.

Original Articles.

ON THE NEURAL PROCESSES UNDERLYING ATTENTION AND VOLITION.¹

BY H. CHARLTON BASTIAN, M.A., M.D., F.R.S.

*Professor of the Principles and Practice of Medicine in University College,
London.*

IN the address which in accordance with custom it will be my duty to submit to you to-night I purpose saying something concerning the neural processes underlying two endowments which are to be found at the root of all our mental and bodily activities. They are endowments naturally obscure and difficult to understand, concerning which great differences of opinion exist both among psychologists and neurologists. I allude to the processes of Attention and Volition. It is still not uncommon for them to be regarded as separate and mysterious 'faculties,' in the old sense, and to find those who hold such views seeking, in the same spirit, to enthrone them in some definite centres in the cerebral cortex. It is the process of attention more especially that has given rise to an enormous amount of discussion during the last quarter-of-a-century. Previous to this period no very great diversity of opinion existed in regard to this mental endowment, or as to the complicity of the processes with which it is associated; but the promulgation of the doctrines of Wundt and Bain in regard to 'feelings of effort' as concomitants of the activity of motor centres or of the outgoing current, followed as it was soon afterwards by the

¹ Being a Presidential Address to the Neurological Society of London.

postulation and supposed location of true 'motor centres' in the cerebral cortex, speedily led to the enunciation of many new views concerning attention, perception, volition and other psychical processes. The brilliancy and importance of the experiments which led to the discovery of the centres in question, combined with the popular view of their interpretation, undoubtedly tended to foster the spread of the parent doctrines.

Now, however, when the fundamental doctrines of Wundt and Bain in regard to the nature and origin of the feelings of effort are believed by many to be absolutely disproved;¹ and when the derivative notion that 'motor centres' exist in the cerebral cortex has also, in the opinion of many, been shown to be almost similarly improbable, it seems an opportune moment to look beyond, and enquire as to the validity of the foundations for some of the new doctrines concerning attention and volition, which are also to be regarded as, in great part, derivatives of the fundamental notions above referred to.

It is clear that the two processes of attention and volition have so much in common that there must be a certain community of interpretation both from the psychological and from the neurological point of view. It is not likely, that is, that one process could be explained correctly from the old and the other from the newer point of view. If these newer views are untenable in the one case, they will be similarly unfit for the explanation of the related psychical process.

Let us first then, as a point of departure, look briefly at the old notions concerning the nature of attention and volition, so far as these were defined by British philosophers up to about the year 1840.

Both Reid and Dugald Stewart speak of attention as though it were an almost purely 'active' process, but James Mill, in his "Analysis of the Phenomena of the Human Mind," regards it rather as a two-sided process, as being both active and passive. Thus, he says,² "the other process

¹ Of Wundt only as regards his supposed 'feelings of innervation'; as he admits that one part of the feeling of effort is of afferent origin.—"Psychologie Physiologique," 1886, t. I., pp. 421 & 445.

² 1829, Vol. II., p. 290.

through which the mind is supposed to influence its trains, is Attention. We seem to have the power of attending or not attending to any object; by which is meant that we can will to attend to it, or not to attend." But on the following page he says—"The state of mind under a pleasurable or painful sensation is such, that we say, the sensation engrosses the mind The phrase engrossing the mind is sometimes exchanged for the word attention. A pleasurable or painful sensation is said to fix the attention of the mind. But if any man tries to satisfy himself what it is to have a painful sensation, and what it is to attend to it, he will find little means of distinguishing them. Having a pleasurable or painful sensation, and attending to it, seem not to be two things, but one and the same thing The feeling is not one thing, the attention another; the feeling and the attention are one and the same thing." Similarly, in regard to other modes of attention, Mill held that, "as in the case of sensation, attending to an interesting idea is merely having it; attention to an indifferent idea is merely associating it with some idea that is interesting." These extracts suffice to show that for James Mill the process of attention was an essentially passive one, and that its apparently active element was only due to the superaddition of Will or Volition.

The view of Sir William Hamilton concerning attention was very similar. That attention is in its essence more of a passive than of an active process is evidently Hamilton's view; since he says:¹—"I think Reid and Stewart incorrect in asserting that attention is only a voluntary act, meaning by the expression voluntary an act of free-will It therefore, appears to me the more correct doctrine to hold that there is no consciousness without attention,—without concentration, but that attention is of three degrees or kinds. The first, a mere vital and irresistible act; the second, an act determined by desire, which, though involuntary, may be resisted by our will; the third, an act determined by a deliberate volition. An act of attention—that is an act of concentration—seems thus necessary to every exertion of consciousness." And previously (p. 237) he had written:—"Attention is conscious-

¹ "Lectures on Metaphysics," (Delivd. 1836-37) Vol. I., p. 247.

ness and something more. It is consciousness voluntarily applied, under its law of limitations, to some determinate object ; it is consciousness concentrated."

The views commonly held in regard to Volition up to the early part of this century were not appreciably different from those put forward by Locke at a much earlier period, who, in the simple language of his time said :¹—" We find in ourselves a Power to begin or to forbear, continue or end the several Actions of our Minds and Motions of our Bodies, barely by a thought or preference of the Mind." No details, however, were offered by him as to the mode in which 'will' was capable of influencing either the actions of our minds or the motions of our bodies. For the first valuable hint as to the details of the latter process we have, indeed, to turn to James Mill, though Hartley had made an attempt in this direction when he said :²—" Of the two sorts of motion, viz., Automatic and Voluntary, the first depends upon Sensation, the last upon Ideas." James Mill's contribution to our knowledge of the subject was, however, much more important. As others had done, he called attention to the fact that a feeling of an emotional order, known as 'desire,' was a necessary prelude to a voluntary movement, but that something else accompanies or immediately follows this emotion of desire—viz., an Idea or Conception of the kind of movement needed for its gratification. He says,³ " We do not undertake to say what physical links are between the Idea and the Contraction, any more than between the Sensation and the Contraction. *The Idea is the last part of the Mental operation.*" This idea of the movement to be executed was declared by Mill to be two-fold in its origin. In reference to this he said :—" There are two ideas very different from one another, to both of which we give the name 'idea of the action.' . . . One is the outward appearance of the action, and is always a very obvious idea." The other is a copy of certain internal sensations, which a few pages previously he had spoken of

¹ "Essay concerning Human Understanding," 1639, Bk. II., Chap. 21, § 5.

² "Observations on Man," 1748, Chap. I., § 3.

³ *Loc. cit.*, Vol. II., p. 266.

generally as "sensations accompanying the movement," and which he also more specifically defined (*loc. cit.*, p. 275) when speaking of the terminal events of a movement as "the contraction of the muscles, with the various sensations which the action upon these organs, and the actions excited in them imply." Of these internal sensations, he says, "from the habit of not attending to them, we have lost the power of attending." And then he adds, "this last (namely the revival of such internal sensations) is by no means an obvious idea. And the mind passes from it so quickly, intent upon the action which is its result, that it is almost always swallowed up in the mass of association. It constitutes, in fact, one of the most remarkable instances of that class of links in a chain, which, how important soever to the existence of the chain, are passed over so rapidly, that the existence of them is hardly ever recognized. . . . This last Idea alone is that upon which the contraction is consequent."

After this brief introduction we will now turn to some of the more modern conceptions as to the nature of attention and volition.

ON THE RELATIONS EXISTING BETWEEN ATTENTION AND VOLITION.

Both attention and volition are in part feelings of a so-called 'active' type; the exercise of each of them being associated with a 'feeling of effort.' Attention is the more primordial endowment; and volition is in part a development therefrom, in which, however, the root process is easily to be detected.

Attention may be directed to impressions made by things without—that is to sensations; or to their revival in idea—the latter mode of activity being known as 'reflection.'

Attention itself is commonly held to mean a "concentration of consciousness." The more our consciousness is narrowed and focussed, as it were, upon some one object or thought, the more developed is that state of mind known as attention. On the other hand, the wider the area of consciousness, at any given moment, (the larger the number of

objects or ideas simultaneously present), the less vivid is our state of consciousness, the less developed is that phase of mind known as attention. This mode of activity is, however, named 'apperception' by Wundt.¹ The entry of an object into the field of vision he speaks of as perception; but when it becomes the object looked at—the central point—there is what he calls apperception, or, as others would say, a process of attention.² With this process, there is always a particular feeling of effort associated. He agrees, in fact, with Fechner in saying that there is always a feeling of tension in the sensorial organs corresponding with an act of external attention, while with an act of internal attention (reflection) there is a similar feeling referred to the scalp, especially in the occipital region.³

But attention varies in its degree of complexity; it is simple or 'spontaneous,' for instance, when we are exposed to some very vivid or novel sensorial impression, or when there is the memorial recall of some extremely painful or pleasurable incident. Attention is usually rendered, under such circumstances, as it were automatically and without effort—and, as Ribot insists, in response to some emotional state.⁴ On the other hand, attention may be initiated in a more complex manner, as in those cases where it seems to be indissolubly associated with an act of will or volition. This is the higher phasis known as 'voluntary attention'; it is the process by which we compel ourselves to observe certain external phenomena, not of absorbing interest in themselves, but for some ulterior purpose; or in which we direct our thoughts upon a given subject with a view to develop our knowledge of its relations.

Voluntary attention appears to be a compound or fusion of the two states, volition and attention. This seems all the more clear when we remember the two spheres of will

¹ According to Prof. Cattell (*Mind*, vol. xiii., p. 438) the word 'apperception' was introduced into philosophy by Leibnitz to denote a spontaneous activity of the mind through which presentations are clearly distinguished. The word was also made use of by Kant and by Herbart, previously to the time when Wundt assigned such a prominent place to it in his psychology.

² *Psychologie physiologique*, 1886, t. II., p. 231.

³ See also Ribot, "The Psychology of Attention," Chicago, 1890, pp. 22, 67-69.

⁴ *Loc. cit.*, pp. 12, 16, and 32.

long ago indicated by Locke. The direction of our thoughts is, in fact, as much a mode of volitional activity as is the production or the control of movements in predetermined modes. Nay, more, it happens that the psychical process preceding the production of all voluntary actions whatsoever differs little, if at all, in its essential characters from that which is comprised in an act of voluntary attention (Wundt). Anteriorly, however, there is in volition another important process; there is more or less of a weighing of motives prompting to this or that kind of action—that is, of deliberation. This deliberation terminates in the dominance of one or other of the motives under consideration, and with this there is generally associated the idea of some action to be performed now or at some future date. This dominance, therefore, makes more vivid the idea of its particular associated movement or series of movements. Coincidentally, if there is to be present action, there is the removal of all ‘inhibition,’ and the ideal movement becomes immediately replaced by the actual movement which it prefigured. As to this latter process Hume said :¹—“ But the means by which this is effected; the energy by which the Will performs so extraordinary an operation; of this we are so far from being immediately conscious, that it must ever escape our diligent enquiry.” To this prognostication we may, I think, fairly demur. Our knowledge of the functions of the Rolandic area, and of its relations through the pyramidal system of fibres with motor centres in the bulb and in the cord, does, in part, redeem us from this reproach of hopeless ignorance.

ATTENTION.

In reference to Attention three problems seem especially worthy of consideration :—[1] as to the essential nature or mode of production of the process of attention; [2] as to the exact relation of attention to motor activity, and as to its intensification or diminution thereby; [3] as to the cerebral seat or mechanisms concerned with this endowment.

¹ “A Treatise of Human Nature,” 1737.

Under one or other of these heads most of the principal difficulties or differences of opinion in regard to attention may be discussed.

1. *As to the Essential Nature of Attention.*

How is it that the limitation in the area, or concentration of consciousness, in which attention seems to consist, is brought about? This is a very difficult problem if the enquiry is confined to the essential neural process itself. In seeking for light on this subject we should, therefore, face it in its simplest form. What happens, for instance, in a case of spontaneous attention to some novel visual impression, when, with all other avenues of sense open, we seem to be aware only of this particular visual impression—all other modes of consciousness seeming to be for the time blotted out? I am not prepared with any new answer to this fundamental question, nor have I been able to find any definite reply thereto in the writings of other neurologists or psychologists.

Some such attempt, it is true, has been made by Maudsley. Speaking of the conditions of consciousness generally, he says:¹—"The persistence for a time of a certain degree of intensity of energy in the ideational circuit would certainly appear to be the condition of consciousness;" and two pages further on he adds, "*Attention* is the arrest of the transformation of energy for a moment—the maintenance of a particular *tension*." This, however inadequate, does afford some sort of an explanation. It gives some account (*a*) of the reason why a particular impression should in certain cases become dominant, and also (*b*) why other impressions should, for the time, be blotted out of consciousness.

As to the first point, we know (*a*) that the adequacy of a given impression to become dominant and all-engrossing depends greatly upon the individual's experiences and tastes, and thus is dependent even more upon the previous activities of other brain elements than upon those now being primarily excited from without. For instance, the effects produced by setting light to a long train of gunpowder would concen-

¹ "The Physiology of Mind," 1876, p. 306.

trate the attention of a savage who had never previously seen such a marvel, very much more than it would concentrate the consciousness of one of ourselves. In the cortical cells excited in us by the impressions derived from the explosion of a train of gunpowder, the molecular activities aroused would, in part, at once well out through a series of commissural fibres to other sensory centres, there to revive related past impressions, so that we should immediately perceive or realize the nature of the external cause. But in the savage, the experience being a novel one, the molecular activity aroused could not at once flow out through such commissural channels; these would not exist; there might consequently be momentary tension in the cells of the visual centres; and (b) the superior intensity of this process might therefore, by comparison, dim or blot out other coexisting but weaker and more familiar impressions—just as, to take a rough example, in coming from bright sunlight into a darkened room we are at first unable to discern anything. Similarly, a pain or a pleasure, but especially the former, may concentrate our consciousness so as to exclude all else, by its mere intensity.

In either of these cases, the novelty, the intensity, the pain, or the pleasure associated with the external impressions, cause them to engross our attention. It is given by no effort of our own; it is, as it were, commanded and no resistance is offered.¹

In the majority of cases, however, where attention comes into play, it does so by virtue of some selective and controlling process, which we ourselves initiate. It is started from within, that is, rather than from without. Such processes

¹ Cycles ("The Progress of Human Intelligence," 1880, p. 138) says: "In cases where Impression chiefly is acting (that is where reminiscence, ratiocination, &c., are not playing much of a part), our attention is quite at the mercy of vividness or intensity * * * In looking at a scene in a merely general way, anything moving attracts us—the flight of a bird, the flashing fall of a cascade, the waving trees, will draw our regard from the still objects; but if among these latter there be one showing colour, it might successfully compete with the things in motion. A shrill cry from another part of the scene might cause us to turn the head wholly away. But if, when gazing at one thing moving, another in the line of vision moved quicker, or if beside one patch of colour another were to show of a fierier hue, shift the eye we must—that is, if we had not some motive to the contrary stirring a volition." This same author makes a very elaborate but obscure attempt to explain the process of Attention from a Neurological standpoint. Still his explanation contains some valuable suggestions (*loc. cit.*, pp. 122-161).

are of various degrees of complexity. But, as Ribot says :¹ —“ In voluntary attention the aim is no longer set by hazard or circumstance ; it is willed, chosen, accepted, or at least submitted to ; it is mainly a question of adapting ourselves to it, and of finding the proper means for maintaining the state ; hence voluntary attention is always accompanied by a certain feeling of effort The birth of voluntary attention, the power of fastening the mind upon non-attractive objects, can only be accomplished by force, under the influence of education It is an instrument that has been perfected—a product of civilization Love of work is a sentiment of purely secondary formation, that goes hand in hand with civilization. And we may note, now, that work is the concrete, the most manifest form of attention. Continuous work is repugnant even to half-civilized tribes.”

All processes of ‘voluntary attention’ are, however, complex phenomena which, as I venture to think, are compounded of volition and attention. Some of the means by which we turn our attention in this or that direction will be referred to in the next section ; while others will be spoken of when we treat of volition itself—which seems to be the most distinctive side of this complex process known as ‘voluntary attention.’

2.—*As to the Relation of Attention to Motor Activity, and its Intensification or Diminution thereby.*

It is the fashion with some at the present day to speak of attention as an essentially ‘motor process.’ This view, however, does not seem to me a just one although, for reasons to be stated, motor activity is inseparably bound up with almost all processes of attention.²

¹ *Loc. cit.*, pp. 35-43.

² Ribot, *loc. cit.*, pp. 8, 25, & 29. This author admits however, that there is no decisive proof as between his view, and that which holds that motor phenomena are simply inseparable elements in attention. Thus he says (*loc. cit.*, p. 60):—“ But the reader will say : ‘ We admit that there are motor elements in perceptions, images, and, to a less degree, in concepts. Still, that does not establish the fact that attention acts upon them, and through them, and that it is a motory mechanism.’ True, upon this point we can cite no observation or experiment that would be decisive. The crucial test would consist in discovering whether a man, deprived of all external and internal motility—and of that alone—would be still capable of attention. But that experiment is not practicable.” Sully, however, says (BRAIN, 1890, p. 155):—“ I think, if we

The cause of this inseparable relationship is, in the first place, the fundamental one, that throughout the animal world expression invariably follows impression ; or, in other words, that by the very constitution of our nervous systems impressions made from without upon nervous ganglion cells, and the molecular motions there liberated, flow towards other groups of ganglion cells, and thence outwards through nervous channels, either diffusely or along definite routes. In this way either generalized or more or less obviously special movements in response are evoked.

In our waking state, when all our senses are to some extent active, a diffuse and almost imperceptible wave of molecular movement is ever welling out to the various muscles of our body, so as to maintain in them the condition known as 'tonus,' together with those persistent, more or less slight, activities needed to maintain the necessary equilibrium between opposing groups of muscles.¹ But, let our sensorial activity be cut off by the supervention of sleep, and immediately there is a diminished reverberation of external impressions through the nervous system ; and there is consequently a diminution of this diffusive outgoing wave, as is at once shown by the nodding head and relaxed limbs of the sleeper.

But, if the states of consciousness which, as a whole, enter into or compose our waking condition are attended by this subdued and generalized outflow of molecular activity making for the whole muscular system, there are also equally fundamental reasons why special excitations of sensorial activity should be associated with special muscular movements—and that, too, as initiatory or accompanying rather than as consecutive phenomena.

The conformation of the sense organs of animals as well as their relation to the external stimuli by which they are

carefully examine our states of mind when we are attending to impressions or ideas, we are in some cases able to distinguish attention as something apart from and independent of muscular activity. Thus, as everybody knows, we may fix the eye on an object and yet not attend to that object. Helmholtz notices the fact that we can attend to an object in the side regions of the field of vision without fixating the object. Where, one may ask, is the motor factor, the process of muscular adjustment in this case ?

¹ See Ribot (*loc. cit.*) p. 26 (2).

excited makes this a necessity. Sounds may come to the ears from all sides; so that movements of the body, of the head and neck, or of the ears themselves are needed for their perfect or better appreciation. The same holds good for visual impressions and visual organs; and how much the sense of touch is heightened by movement is familiar to all. Taste is also intensified by certain movements, and, though to a still smaller extent, the sense of smell. Throughout the whole animal series, and as long as animals have existed, there must always have been this inseparable relation between the several modes of sensorial activity and movements. Such movements are, as we have seen, either more or less permissive of the sensorial activity, as when the head is turned in a particular direction; or they go to develop or intensify the impressions that can be received, as when movements of the eyes, ears, or hands take place with the effect of vastly augmenting the range or the intensity of the respective visual, auditory, or tactile impressions.¹

What wonder then that in association, for instance, with visual or auditory impressions there should have grown up an inseparable motor response of a more special kind, to wit, a turning of the head and eyes in the direction of the sight or sound, together with a fixed attitude of the body generally.

That there should be an 'attitude of attention' is, as we see, perfectly natural; just as it is perfectly natural that with different emotions there should be different special

¹ 'Adjustment of attention' to its objects is commonly spoken of as though it were a motor process. Wundt does so, for instance, because of the existence of sensations of tension referred to the corresponding sense organs. In 'pre-attention' or 'expectant attention,' such sensations may, it is true, come from merely permissive muscular actions in the sense organs. But if reaction time is diminished when the nature of the impression is known beforehand, and still further when the time of its occurrence is also known; such adjustment is surely in great part sensory and dependent upon the fact that an image or idea of the event foreseen is evoked, and evoked at exactly the right time, so that, as Ribot says (*loc. cit.* p. 74), "the real event is but the re-inforcement of the representation already existing." The supposition made by Bain and others that mere ideal recollection of perceptions involves a feeling of tension in the corresponding sense organ is very questionably true. Such feelings of tension or effort (in accordance with the view that they are of afferent origin) would require distinct contractions of muscles for their generation. But obvious contractions of muscles are mostly absent in such cases. And no one has peripherally initiated feelings of effort from mere "nascent" excitations of muscles, stopping short of producing actual contractions.

motor accompaniments. But because of these inseparable motor accompaniments, it seems to me we might almost as reasonably say that emotion is an essentially motor process as make such an affirmation concerning attention.

Each of these mental processes is, I believe, especially concerned, in different modes, with the activity of sensory elements, although a special welling over of molecular activity into related motor mechanisms is also almost inseparable from the activity of each of them. In other words, we have to do both in emotion and in attention with sensori-motor processes, though, in my opinion, the ganglionic elements concerned with the motor side of this activity lie altogether outside the cerebral hemispheres, just as the activity of such motor mechanisms lies altogether outside the sphere of consciousness.¹

The question of the direction of attention, or the degree to which it may be intensified or diminished is one which really pertains to the subject of volition, as it only comes

¹ Thus, to say, as Prof. James does ("Princip. of Psychol.," 1890, vol. i., p. 30):—"Ideas of sensation, ideas of motion are, on the other hand, the elementary factors out of which the mind is built up," is quite in accordance with my own beliefs. We have in the cerebral cortex an extended register of two kinds of sensory impressions—those which primarily *incite* to movement together with other sensory impressions (kinaesthetic) resulting from such movements and constituting the subsequent *guides* for the execution of similar movements. Just as the different kinds of sensory centres belonging to the first set are brought into intimate relation with one another by means of commissural fibres, so are the kinaesthetic centres similarly connected with each of the other sensory registers. The statement quoted above is, however, quite a different thing from saying as Hughlings Jackson ("Clin. and Phys. Researches on the Nervous System," 1876, pp. xx-xxxvii.) has done with much insistence that "mental operations in the last analysis must be merely the subjective side of sensory and motor substrata"; or from saying as Ribot ("Les mouv. et leur import. psychol." in *Revue philosophique*, Dec., 1879) does that we have such things as "motor ideas," that "movement and sensation are the stuff of which our mental life is composed," or that "at the root of our mental life, everywhere and always there are movements." I should agree to the last proposition if "ideas of movement" were substituted for "movements." All the other modes of expression imply that there are motor centres in the cortex, and that the activity of motor centres carries with it a subjective phasis, both of which positions I, in common with James, Münsterberg and others, believe to be erroneous. The "idea of the movement," apart from its visual component, is an image of a body of ingoing impressions caused by the movement itself, and the registering centre for these impressions has precisely the same claim as the visual and the auditory to be regarded as a sensory centre. This is the view also now held by Ribot, though formerly, as we have seen, he used language seeming to imply that ideas of movement are revived in motor centres ("Psychol. of Attention," p. 54). His real view is explicitly stated, however, on pp. 26, 27, 66 and 67.

into play when 'voluntary attention' is being exercised. This subject will, therefore, be referred to when I speak of the mode by which our thoughts are controlled or concentrated in this or that direction.

3. *As to the Cerebral Seat or Nervous Mechanisms concerned with Attention.*

In his recent very interesting paper, read before this Society, on "The Psychophysical Process in Attention," Sully says: "In England as well as in Germany the question of the precise region of the cortex involved in the process of attention has been the subject of considerable discussion."¹ It is well known, also, that Ferrier localises the "faculty of attention" in the frontal lobes. His most recent statement on the subject is this. Speaking of the effects caused by removal of the frontal lobes, he says:² "I have also observed (and my observations have been confirmed by Hitzig and Goltz) a noteworthy psychical defect—a defect which I have endeavoured to correlate with the inability to look at, or direct the gaze towards, objects which do not spontaneously fall within the field of vision. It is a form of mental degradation which appears to me to depend on the loss of the faculty of attention, and my hypothesis is that the power of attention is intimately related to the volitional movements of the head and eyes." This latter view Ferrier

¹ BRAIN, 1890, p. 147. Sully, in fact, seems to suppose that we have to look in the main for some "higher motor centre" as being specially concerned with the process of attention. He says elsewhere ("Outlines of Psychology," p. 77): "The fact that attention is an act of the mind would suggest that its nervous concomitants are certain processes in those nerve centres which we know to be more especially concerned with movement and action. This conjecture is borne out by the fact that the act of attention is commonly accompanied by muscular contractions"—referring here to the movements of the head and eyes and the attitude of the body generally during acts of attention. Again, further on, he says (*loc. cit.* p. 661): "In the control of movement and of feeling, nervous influence appears to pass from the higher motor centres (including those of attention) to the lower motor centres from which this process of innervation concerned in the impulsive or emotional movement sets out." Still Sully has since contended (BRAIN, 1890, p. 157) that attention "cannot wholly be resolved into a motor phenomenon." He adds: "The relation of the two is, I suspect, very similar to that which obtains between an emotion and the several sensory and motor phenomena which accompany it." This latter conclusion may be admitted as correct; but the holding of such a view is surely not in accordance with the language previously quoted.

² "Cerebral Localization," 1890, p. 151.

puts elsewhere in the following words:¹—"The faculty of attention with all that it implies in the sphere of intellectual operations, must be intimately related to the volitional control of the head and eyes in association with the centres of visual perception and ideation."

No attempts at the localisation of attention are made by Bain. He, in fact, gives no details concerning attention, or as to the way in which it is brought about.

Wundt, however, does postulate a distinct organ for apperception (attention) which he also is inclined to localise in the convolutions of the frontal lobe.² But it should be said that his theories concerning apperception, its localisation, and the modes of cerebral activity with which it is associated are entirely speculative and fanciful.³ He postulates the existence of this centre of apperception, to which, as he imagines, all sensory impressions are sent in duplicate (by way of the cerebellum), and from which again he postulates the issue of one set of fibres to each of the cortical sensory centres, together with another set to each of the supposed motor centres of the cortex. What happens when attention is aroused by some external object is, according to Wundt, as follows:⁴—The first effect is the production of an image of an intuitive or imaginary order, not sharply defined, but as it would be if much to the side of a visual field. This, Wundt regards as the result of the stimulus upon the cortical visual centres, but he adds: "The sensorial irritation is at the same time transmitted to the central territory of apperception. Thence the stimulus follows a double route and directs itself (1) backwards towards the sensory territory with the effect of reinforcing the image; and (2) towards the motor centres which innervate the voluntary muscles, as a result of which there are necessarily produced those muscular tensions that assist to constitute the feelings of effort accompanying attention, and which react in their turn upon attention—reinforcing it in conformity with the

¹ "Functions of the Brain," 2nd Ed., 1886, p. 264.

² *Loc. cit.* t. I., p. 245, and fig. 65.

³ See art. by Bain in *Mind*, 1887, pp. 174-8.

⁴ *Loc. cit.* t. II., p. 236.

law that associated feelings lend one another a mutual support."

With most of these statements and points of view I have myself no sympathy. As will have been seen, I take a totally different view as to the relations of the centres concerned with the turning of the head and eyes to the process of attention from that adopted by Ferrier, nor can I regard attention, whether called by its own name or by that of 'apperception,' as a 'faculty' which, somewhat in the old phrenological sense, is to be definitely localised in this or that portion of the cortex; and least of all could I think, if it is to be topographically localised, that we should place this essentially sensorial endowment in that region of the brain which may, truly enough, be concerned with the production of one of its commonest motor accompaniments or reactions. The reason why there must necessarily be such motor reactions and accompaniments is, as I have said, dependent in part upon the constitution of our nervous systems, and in part upon the fact that we are compelled to react by turning the head and eyes in this or that direction, because from all sides visual and auditory stimuli may come to us. The relation between these particular movements of the head and eyes and attention do not, therefore, convince me that they are other than almost necessary results of sensorial activity. On the other hand, I should not think of attempting to localise the process known as attention in any one definite part of the brain, but should regard it as having its loci in cell and fibre mechanisms in each one of the cortical sensorial centres—that is, as being concerned with mechanisms scattered all over the cortex, according as we are, with more or less predominance, attentive to visual, auditory, tactile, olfactory, gustatory, or kinaesthetic impressions.

VOLITION.

As already intimated the sphere of what is called Volition is commonly believed to be two-fold. Thus, one form of such activity is supposed to exist which manifests itself in the control or guidance of our thoughts; and another in the pro-

duction of muscular movements. These two processes correspond with what Wundt terms the "internal and the external activity" of Will. What really happens in these two cases we must now consider.

In the first place it is necessary, here also, to get rid of the erroneous notion that under the head of Will or Volition there is to be included a distinct 'faculty' having a cerebral localisation of its own in some part of the cortex—an imaginary 'faculty' acting as a well-spring of power, isolated and mysterious in its origin. Still less can we believe that Will is "a species of spiritual rudder distinct from and superior to the automatic forces of the brain."¹

According to Bain the external and the internal modes of activity of will are essentially similar; he holds that in each case the fundamental action is through motor centres and muscles, and his view has been adopted by Ribot and others.²

Bain says:³—"I look upon volition as existing only in connection with the active organs, that is, with the muscular system. Even in the sphere of thought the limitation holds." For Wundt, James and others, also, any difference that might be thought to exist between the two processes is more apparent than real. They, however, differing from Bain, regard as fundamental the nervous processes that occur anterior to the actual incitation to the movement—holding, as they do, that the activity of muscle is a non-essential and mere physical addendum to the volition itself. As Wundt points out, in certain cases of paralysis the will to move may be there and made—even though the movement does not follow.⁴

¹ *The Spectator*, June 6th, 1891, p. 793.

² *Loc. cit.*, p. 51.

³ "The Senses and the Intellect," 3rd Ed., p. 559.

⁴ As to the psychological characteristics of the two modes of volitional activity (internal and external) Wundt (*loc. cit.* t. II., p. 445) contends that there is a feeling of internal activity or effort associated with each of them, but in cases of the latter type where movements follow, he says, "this feeling receives here a characteristic colouration because it becomes fused with the sensations of the movement itself, so as to constitute an inseparable complexus." The birth of this fusion becomes, therefore, for Wundt the fundamental basis by which 'external voluntary activity' is to be distinguished psychologically from 'internal voluntary activity.'

*The Nature and Extent of Voluntary Power over the Control
and Direction of our Thoughts.*

That we have some power of controlling or directing the succession of our thoughts is generally recognised, and confirmed by the consciousness of each one of us. It seems obvious, also, that this aptitude may not only be improved by practice, but that it is naturally much better developed in some persons than in others—and especially in those whose powers of attention are strong.

In all such processes of direction or control of thought we have the manifestation of the highest phase of attention—that known as ‘voluntary attention’—the consideration of which was previously postponed till the present stage of our enquiry was reached; so that what is now to be said will bear as much upon the subject of voluntary attention as upon volition proper.¹

I must take it as commonly admitted that the succession of our thoughts depends upon what is known as the ‘association of ideas’; and that these associations are but reflexes of the sequences and coexistences obtaining among the various sensorial impressions to which we are and have been from moment to moment exposed throughout our whole lives. Thus the mind of man is, as Leibnitz called it, a ‘mirror of the world,’ so far as it is revealed to each one of us.

Again, it is well understood that for the purpose of influencing or altering trains of association, the power of direct impressions is more potent than that of mere remembered impressions. Consequently, when we are in the midst of a train of abstract thought, any vivid impression coming to us

¹ What is now to be said may constitute an answer, in fact, to those who still believe in the spiritual conception of Will. Thus a writer in *The Spectator* (June 6, 1891, p. 789) has recently said that “voluntary or scientific attention” is, “an act of pure *will*, meaning by will, of course, not that which is the resultant of pre-existent impulses and desires, but that self-caused effort by which scientific attention is distinguished from all such acts of involuntary attention We know nothing if we do not know that we ourselves cause for ourselves all such acts of attention, and that science of all kinds is the organised result of such acts As we said last week, ‘will’ is a word of supererogation, it is a will-o’-the-wisp which has no pretence for existing at all, if it represents nothing but a resultant of desires.” Yet that this latter view is true, we shall now endeavour to show.

from without will tend to arouse associations of its own, and thus disturb our previous trains of mere reminiscence.

If the subject on which we are thinking is associated with external observation, the one or two senses concerned are of course active and receptive, and the vivid impressions that we receive through them suffice to fix our attention and control our thinking processes. If we wish to divert our attention from either of these vivid sensations, so as to be enabled to follow some other line of thought we can avert our eyes, or close our ears, so as to cut off the disturbing impressions. In abstract thought we often naturally close our eyes, court silence, and keep perfectly still, so as to have no externally derived impressions of any kind disturbing our trains of association.

We are concerned in all such cases with mere reminiscence or memorial recall of past impressions, together with intellectual activities related thereto. During such a process portions of almost the whole of the cortex may be maintained in a condition of full or sub-conscious activity as, with "the rapidity of thought," molecular notions pass along countless definite lines or associating channels between this and that group of ganglion cells. The nervous activity tends in each case to pass most and with greatest ease along those channels which have hitherto been most frequently traversed—taking the routes, that is, which custom has rendered "lines of least resistance." As an able writer says: "In this way trains of thought of any length may be excited; until the original nervous activity either emerges by some form of expression into the outer world, or becomes absorbed in the stronger current of a fresh direct sensation."¹

Thus it is found that our thoughts invariably follow one another by the laws of habit or association (these being, as I have said, a reflex of external co-existences and sequences), the most vivid sensation or idea for the time being ever filling our consciousness and rousing, through associational channels, related ideas and concepts. So that the continuance for a time of any one of them merely proves that during the

¹ Barret, "Physical Ethics," 1869, p. 315.

period of such continuance there is no other strong enough to drive it out.

If, therefore, our thoughts succeed one another in this way according to definite laws, it may be asked (a) what is the province of volition or will in regard to their direction, and (b) by what means can any such influence be exerted?

(a) As to the first question I believe nothing more definite than what Barret expresses in the following passage can be said:¹ "We may, indeed, if we like, give to those sequences in which one train of thought *supersedes* another the name of 'will,' as distinguished from those which are regular members of one continued train; but their nature and foundation must be the same in the one case as in the other; for the second train must have some origin like the first, and can only conquer by its superior force." It comes, in fact, to this, that our thoughts invariably occur in accordance with the 'laws of association'; but that in some cases, changes in their order, or a persistence in the same order, is said to be a result of 'will.' It is well known that Wundt and others assert the need of something beyond the mere association of ideas as being required for a scientific understanding of the so-called 'active' powers of mind, and that it was with this end in view that he postulated the existence of his so-called faculty of apperception, carrying with it activities of its own apart from the ordinary laws of the 'association of ideas.' Münsterberg has of late conducted two most important and skilful experimental investigations bearing upon the question of the existence or not of any such need. After a careful analysis of these researches Croom Robertson sums up the impressions they have produced in the following manner:² "Taken together the two researches in their different way certainly point to one conclusion—that there is no such difference between so-called voluntary and involuntary intellection as Wundt's apperception theory (or any other like it) would make out."

(b) It remains then to consider how this superseding of one train by another, or continuance of an old train in spite

¹ *Loc. cit.*, p. 142.

² *Mind*, 1890, p. 242. F. H. Bradley also expresses strong opposition to Wundt's doctrine of apperception (see *Mind*, 1887, p. 366).

of a tendency to wander, is or can be brought about; in order to indicate the kind of means by which what is called 'will' acts in directing or controlling the course of our thoughts. On this subject a few hints only can be given, concerning the different methods known to be adequate for the attainment of such ends.

(1) In the first place it should be stated that there is a sort of antagonism between the mental activity of mere reminiscence, and that which is set on foot or maintained by external impressions—for the reason that the same central elements are called into activity in the two cases. Consequently, when any very interesting train of thought is being carried out we may become for the time 'self-absorbed,' and, during this period, much less receptive of external impressions, so long as they are not very intense. Moreover, when so engaged we always try to guard ourselves from the possible disturbing influence of external impressions of all kinds.

(2) Of the two opposing processes that which is externally derived (impressional activity) is decidedly more vivid and potent than that which is internally initiated (reflective activity); and it is this fact that enables us at any time (*a*) to interrupt and supersede a train of thought proceeding in accordance with the laws of association; or, on the other hand (*β*) to maintain it when we so desire.

In the one case, in order (*a*) to break in upon a train of association we may call into action some vivid sensorial impression, and we may repeat this operation till we have finally displaced the old train of thought—often simultaneously seeking the aid of bodily movements of one or other kind, which are potent in this direction by reason of the kinæsthetic impressions that ensue, and the new trains of association that may be initiated thereby.

While, in the other case, where (*β*) we seek to maintain some train of thought in spite of a tendency to wander to other subjects, we call in the aid of language. That is, we repeat to ourselves some words essentially related to the subject of our thoughts, or we may sum up the stage at which we have arrived in words, thereby reinforcing the existing

associational activity to which our attention is being given, through the mixed auditory and kinæsthetic impressions produced. How very influential this mode of control is may be gathered from the added power of this kind that we obtain when we call into play the aid of writing, and thus help to maintain desired trains of reminiscence by means of additional reinforcing sense impressions, of the visual and kinæsthetic order. Most of us must be familiar with the much greater ease with which we keep our thoughts along certain lines, and even develop them, when we are committing them to writing, as compared with what happens when we do not call into play this extra aid.

Thus, whether we seek to alter, or to maintain and develop, any particular line of thought, we call up as many new impressions as possible—of unlike kind when we wish to disturb or break in upon a previous line of reminiscence; but of like or of related kind where we desire to strengthen and develop the previous associations upon which we have been intent.

What has been said above might seem at first sight to lend support to the views expressed by Bain,¹ that "in all voluntary control of the thinking trains there is a muscular intervention" of a direct kind, so that, as he contends² "the retention of an idea in the mind is operated by voluntary muscles." This, however, cannot be admitted. Will operates, as we have seen, through the muscles only in an indirect way—that is, the muscular actions to which we resort give rise to ingoing impressions, and it is these which, according to their nature, may break up or may reinforce pre-existing trains of association. That is, there is a superseding or a strengthening of previous lines of thought, but in either case the effect still occurs in strict accordance with the laws of association.

It is perfectly true, therefore, that the excitation of muscles (more especially those of our sense organs or those concerned with speech) is an essential preliminary to the guidance or control of our thought processes, though this guidance or control seems not to be brought about as Bain

¹ "Senses and Intellect," 3rd Ed., p. 421.

² *Loc. cit.*, p. 370.

would have us believe, or in such a way as to justify the language which he employs.

It must be borne in mind, in fact, that this voluntary muscular activity itself occurs as a sequence of one of the links in the association of ideas; and that the resulting control or change in the direction of our thoughts is brought about by the sensory results of the movements thus induced—that is, by auditory or visual in concert with kinæsthetic impressions reaching the brain, and again, according to their nature, and by the ordinary laws of association, helping either to change or maintain our previous line of thought.¹

The Nature and Extent of Voluntary Power over the Movements of our Bodies.

The occasions for the exercise of voluntary movements of all kinds spring up, as I have pointed out, as ordinary links in the chain formed by the association of our ideas.

Sometimes simple voluntary movements occur with all the rapidity, ease, and lack of effort that characterize a so-called ideo-motor act, as when I resolve on certain occasions to sit or stand; to raise a glass of water to my lips when I am thirsty; or to eat when I am hungry. The initiatory causes of such actions are only a very little more complicated than those of simple spinal reflexes; the principal difference consisting, as Hartley long ago pointed out, in the fact that instead of being started by an external impression, they are initiated by ideas, although they may be more remotely prompted by internal or external impressions.²

¹ The view of Prof. Bain concerning the direct intervention of muscles in the control of our thoughts, is of course intimately dependent upon his particular view as to feelings of movement being in great part 'concomitants of the outgoing current' and being realized in motor centres. This naturally implies ideal recall in the same centres. Ferrier, strangely enough, though rejecting Bain's views in these latter respects, still adopts his derivative doctrine as to the scope of volition and the mode in which it effects control over our thoughts ("Functions of the Brain," 2nd Ed., p. 461). Ribot seems to show a similar inconsistency. He rejects Bain's notion concerning the mode of registration of muscular sense impressions ("Attention," pp. 66 & 71) and yet adopts his derivative views concerning the mode in which our thoughts are directed (*loc. cit.*, p. 64).

² There is, in fact, no line of demarcation to be found between the cerebral reflexes known as 'ideo-motor' acts, and simple or very familiar voluntary acts. It is often impossible to say which is the more appropriate designation for such acts. In regard to ideo-motor actions Müller said long

At other times our voluntary actions only take place after more or less complicated previous processes of deliberation, or weighing of motives, and with much or little sense of effort. As W. James says:¹—"In the dentist's chair, one idea is that of the manliness of enduring the pain, the other is that of its intolerable character. We assent to the manliness, saying 'let it be the reality,' and behold it becomes so, though with a mental effort exactly proportionate to the sensitiveness of our nerves. To the sailor on the wreck, one idea is that of the sore hands and the nameless aching exhaustion of his whole frame which pumping involves. The other is that of a hungry sea engulfing him. He says, 'rather the former,' and it becomes reality in spite of the inhibiting influence of the comparatively luxurious sensations of the spot in which he for the moment lies. . . . But in other cases both alternatives are images of mixed good and evil. Whatever is done has to be done against some inhibitory agency, whether of intrinsic unpleasantness in the doing, or of represented odiousness of the doing's fruits; the fiat has to occur against resistance. Volition then comes hand in hand with the sentiment of effort. . . . What does this effort seem to do? To bring the decisive volition. What is this Volition? The stable victory of an idea although it may be disagreeable, the permanent suppression of an idea although it may be immediately and urgently pleasant."

At the same time that ideas and motives are in this way in conflict, we have half-aroused or nascent images revived in other sensory centres representing the alternative actions or movements that would naturally be associated with this or that motive were it allowed to be operative. But it is only when one of these motives proves victorious—proves stronger, that is, than its rival—that all resistance is removed; we consent to its supremacy, and the result is

ago, "The idea of a particular motion, determines a current of nervous action towards the necessary muscles, and gives rise to the motion independently of the will." At times we may have the production of actions generally regarded as voluntary, taking place in this simple reflex fashion, and by aid of the same nervous mechanisms. This is also pointed out by Wundt (*loc. cit.* t. I., p. 443).

¹ "The Feeling of Effort," 1880, pp. 23 and 22.

that the revived image of its associated action becomes the immediate incitor of the real action which it foreshadowed.¹

Thus it happens that, in order to bring about some present or prospective pleasure, or the avoidance of some present or prospective pain, we feel a desire to perform certain definite movements, the ideas or images of such movements being aroused, as James Mill originally pointed out, as almost sub-conscious sensorial links in the chain of our thought processes.

From what has hitherto been said two important conclusions may be drawn. First, that a 'sense of effort' is associated with the conflict of ideas and motives which precedes the ascendancy of one of them; and that this sense of effort must, therefore, be an appanage of the activity of sensory centres and their annexes by the aid of which intellectual processes are carried on. There is no good reason for believing that the action of muscles has anything to do with the generation of this particular 'sense of effort'.²

Secondly, that the 'act of willing' any particular movement consists essentially in a consent (after the balancing of reasons that may exist for or against) to the occurrence of such a movement; the movement itself being at the time mentally prefigured by certain revived sensations—such revival representing, as James Mill said long ago, "the last part of the mental operation." The occurrence or not of the movement is to a certain extent an accident, and one which, when it occurs, lies altogether outside the mental process itself.

Let us look then now a little closer at these last links in the chain of association—that is at "the last part of the

¹ See Herbert Spencer, "Principles of Psychology," 1870, vol. I, p. 496.

² Another source of the sense of effort is intimately associated with the activity of our muscles. Its origin is to be found in the ingoing sensory impressions of various kinds whose termini and seats of registration are the kinæsthetic centres in each cerebral hemisphere. This is fully admitted by Wundt, though not by Bain, to be one source of the 'sense of effort.' Some confusion has, I think, lately been introduced into this already sufficiently complex subject by Waller in a paper entitled "The Sense of Effort: an Objective Study" (BRAIN, 1891, pp. 179-249). This title seems to me peculiarly unfortunate, in more ways than one. Interesting and carefully worked out as many of the experiments are, they do not seem to me to touch the question of the origin of the 'sense of effort' as the term is understood

mental operation" which leads on to the performance of a voluntary movement. It consists in a revival of the idea or conception of the movement to be executed. This idea has always at least a two-fold basis, though the actual production of the movement often requires a three-fold excitation of sensory centres in immediate succession, as I shall presently show.

For limb and trunk movements the idea is composed of revived visual and kinæsthetic impressions which have previously been received during the execution of similar movements. The reawakened activity of these sensory centres affords the necessary stimulus and guidance for the reproduction of the movement—the molecular actions associated with their excitation evoke, that is, the related suitable activity of motor centres in the spinal cord. So that as W. James puts it, "every representation of a motion awakens the actual motion which is its object, unless inhibited by some antagonistic representation simultaneously present to the mind." The same kind of thing happens in regard to speech movements, only here we have the reawakened activity of auditory in conjunction with kinæsthetic centres

by psychologists. I find therein no evidence to shake my opinions on this subject, and, moreover, find modes of stating my views to which I cannot assent. I hold that the functioning of muscle itself, of motor nerves, and of motor centres, are mere physiological processes devoid of subjective accompaniments; yet these are the structures in which Waller looks for after-effects that he assumes to be "the same as or similar to, or parallel with, the material substrata of the concomitant sensificatory phenomena" (p. 192). That is to say he seeks for the residual effects of what he says is variously termed "sense of effort," "muscular sense," "sense of movement," or "sense of innervation," in points along the track ABM of Fig. 1 (p. 31) with the whole of which, excepting one part, I say they have nothing directly to do. He seems to think that I consider the peripheral changes in this track as of most importance; while, as matter of fact, I say that the peripheral changes in muscle which he investigates, have nothing directly to do with any of these psychical phenomena. I hold that the contraction of muscles forms the occasion merely for the birth of a series of ingoing impressions starting from the peripheral ramifications of afferent nerves in muscles, joints, skin, &c., which are conveyed along the route MCA, and are consciously realized in the centre A. He calls this latter an efferent or motor centre, and thinks the psychical events he names above are dependent mainly upon its molecular activity. This is exactly what I have long since said, with the important exception, that I maintain the centre in question to be an afferent one (see p. 30). I find in Waller's paper no new independent evidence of any kind bearing upon this latter problem; and much in his mode of presenting the old evidence to which I cannot assent. His paper, in my opinion, must be taken solely as a contribution to what is known as "physiological fatigue"—as he, in fact, would seem to indicate by his sub-title.

starting the stimuli needful for calling into activity the proper motor centres for speech in the bulb.

I would only add here that this view of James Mill¹ is also the doctrine taught as to the mode of production of voluntary movements by Lotze² and by Herbert Spencer;³ that it has been advocated independently by W. James and myself since 1880; and later by Münsterberg,⁴ Horsley⁵ and others.

I have always considered that in the conjoint sensory

¹ *Ante*, p. 4.

² "Medicinische Psychologie," 1852.

³ "Principles of Psychology," 1st ed., 1855, p. 613, and 2nd ed., 1870, vol. I., p. 496.

⁴ Münsterberg's theory (see *Mind*, 1888, p. 463) concerning the 'muscular sense' and its relation to voluntary movements is identically the same as that published independently by Prof. James and myself in 1880, though this seems not to have been recognised by Croom Robertson, since he speaks (*Mind*, 1890, p. 525) of it "as mediating between the opposed theories that have thus far occupied the ground." His attempt in the same place to claim some justification for the view of Wundt and Bain seems to me not very successful. Thus he says (p. 527): "The difference on the afferent side of the system between sensation and representative image is allowed to be one that depends only or, at least, mainly, upon degree of excitation: this being (normally) greater when determined from the periphery. How, then, should there not be a corresponding difference of representative and presentative experience on the *efferent* side when the cerebral process in one case is not, and in the other is, effective in producing overt muscular contraction?" My answer to this is twofold. In the first place, I say that the centres to which his query refers do not stand on the *efferent* side of the nervous system; they (*i.e.*, the kinæsthetic centres) are the cortical termini for a definite class of impressions, and, like the visual and the auditory, are true afferent centres (see p. 32). And, secondly, I would say that his analogical argument sets up a claim for subjective processes in association with the functioning of efferent centres, whilst I, in common with James, Münsterberg and others, maintain that no independent evidence exists to show that such functioning is ever by itself associated with a subjective phasis. His eyes are open to the possibility of this latter retort. He sees, in fact, that his analogy would be rendered nugatory by the denial of subjective phenomena in association with the functioning of motor centres—but he omits to mention that this heresy is maintained by James and Münsterberg as well as by myself. I fail to see, moreover, the cogency of his objections to the use of the term 'kinæsthesis,' and its derivatives. I would remind him that the term 'muscle sensation,' of which he seems to approve as an alternative, refers to only one element entering into the complexus of sensations produced in us whenever we make movements; and that the various components of this complexus are always simultaneously subject to revival as guides for future movements. Surely, if groups of impressions like this always occur together, and are always revived together for the carrying out of important physiological processes, that is a sufficient justification for speaking of them under some common name. And if we bear in mind the fact that such impressions, like all others, are when realized, as Croom Robertson points out, always "overlaid by representation," it may be seen that we are thereby enabled almost intuitively to interpret them as meaning movements of this or that part of our body. How could we, then, better convey the desired meaning than by speaking of them as 'kinæsthetic' impressions?

⁵ *The Nineteenth Century*, June, 1891, p. 857.

revivals, or 'ideas of movement,' the visual or the auditory impression, as the case may be, is the first to be revived, and that the renewed activity in one or other of these centres is passed on through associating fibres to functionally related portions of the kinæsthetic centres. The activity of these latter centres seems to me to be almost if not always evoked in this secondary manner, although for the actual production of the suitable movements the functioning of the kinæsthetic centres is all important. They are situated in the cortex at what has been termed the "bend of the stream"—they are indeed, the last portions of the cortex to be aroused in the performance of voluntary movements, and from them actually issue the fibres (*viz.*, those of the pyramidal tract), which convey the appropriate incitations to the real motor centres situated in the bulb and in the spinal cord.¹ Here then, as elsewhere, motor centres produce (through the intermediation of nerves and muscles) movements which are qualified as to nature, range, and force by the precise nature of the stimuli which they receive from sensory centres.²

How all important these sensory activities are for the production of voluntary movements is well shown in many cases of brain disease leading to speech defects. Thus, I have had under observation from time to time since 1878 a man who then became paralysed on the right side, and whose powers of expression by speech and writing were at the same

¹ I purposely, for the sake of simplifying the problems under discussion, omit all reference here to the co-operative action of the cerebellum in the actual production of movements.

² I am sorry that Dr. Ferrier has again not taken the trouble to state my views correctly. In his most recently published work he quite misrepresents them by stating that I have taught that the kinæsthetic centres are aroused as "independent centres of activity, irrespective of the stimuli from the sensory centres of the cortex" ("Cerebral Localization," 1890, p. 147). This supposed view of mine he then proceeds to refute by quoting the experiments of Marique, confirmed by Exner and Paneth, to the effect that "when the motor [kinæsthetic] centres have been completely isolated, by section of the fibres which associate them with the sensory centres of the cortex, paralysis results of precisely the same character as that which occurs when they are completely extirpated. Marique proved that the same contractions were obtainable on electrical irritation of the respective centres after, as before, isolation, showing that they still retained their excitability and connection with the pyramidal tracts." So far from being opposed to my views, it will be seen that these experimental results are in exact accordance with what I have always said in regard to the mode in which the activity of the kinæsthetic centres is evoked. See, for instance, "The Brain as an Organ of Mind," 1880, p. 593, and elsewhere; also *BRAIN*, Ap. 1887, pp. 7, 57, and elsewhere.

time disordered in a remarkable manner.¹ The principal persistent defect in this man has been a damage to the commissural or associating fibres connecting the visual and the auditory word centres, so that he is unable to perform any movements that require for their initiation and guidance the successive conjoint activity of these centres: showing how absolutely dependent our thought processes are upon the integrity of the associational paths. Thus, a blockage occurred twelve years ago in the lines of communication between the auditory and the visual centres; and as a consequence this man cannot name objects at sight, or read aloud words or even letters; though he recognises them perfectly, can read to himself with comprehension, and can repeat names of things, or words of any kind, immediately that he hears them. Again, though he has now learned to write with the left hand, and he can without hesitation copy in a rude way any words that are written on the paper before him, he cannot write a single word spontaneously or from dictation—not even a letter. The auditory stimulus will not pass along the blocked track,² so that there is no means of rousing the related portion of the visual centre or, consequently, of the kinæsthetic centre. Along this route the idea of writing movements cannot be revived, and as a result, the man cannot make even an attempt to form a single letter. But his puzzled look and passive state gives place to one of pleasure when he sees the word written, and he immediately proceeds to copy it; just as, in the previous trial, his eager tentative look at the word he seeks but is unable to articulate, is followed, the instant he hears it pronounced and thus receives an auditory revival, by its correct utterance. A block in the commissures connecting the auditory with the related glosso-kinæsthetic centre, would similarly prevent reading or naming at sight, as it would also prevent all spontaneous speech; and a block between the visual and the related cheiro-kinæsthetic centre

¹ His case is detailed in "The Brain as an Organ of Mind," p. 642.

² In silent thought, and therefore for spontaneous writing also, the first memory of the word to be revived is, I believe, in the great majority of persons, that which is registered in the auditory centre.

would similarly render writing from dictation, or spontaneous writing, impossible.¹

Here, then, we get at the very roots of Will. The sources of the power employed for arousing the appropriate contractions of our muscles is to be found in the molecular activities issuing from these sensory centres. This is shown by the fact that we have in such cases as I have above referred to, persons 'willing' but unable to successfully execute certain speech movements at the instigation of appropriate visual impressions, though they retain the power of producing such movements in response to appropriate auditory stimuli; and, on the other hand, persons unable to produce writing movements at the instigation of auditory stimuli, which they are able at once to evoke at the bidding of visual impressions.

The same kind of conclusion, as to the source of the energy employed in the production of muscular movements generally, has been forced upon Gotch and Horsley, during their recent important electrical investigations concerning the excitability of different regions in the brain and spinal cord, which in 1890 formed the subject of the Croonian Lecture before the Royal Society.² I note, however, that even these investigators speak of the cerebral kinæsthetic centres as being on the "efferent" side of the nervous system.³ This seems to me an erroneous interpretation. They are, I think, afferent centres in every way analogous to the afferent centres situated in the spinal cord.⁴ The so-called "motor centres" of the cortex were not, of course, originally supposed to be termini for afferent impressions; when first discovered they were said, and they are still maintained by Ferrier to be, true "motor centres." Now, however, in spite of the different interpretation which has been given of their functions, many still cling to the belief that the centres in the Rolandic area must be motor centres because internuncial fibres connect them with the real motor centres in the bulb and spinal cord—and because, therefore, 'motor incitations' must pass along

¹ See a paper "On Different Kinds of Aphasia," *Brit. Med. Journ.*, Oct. 29th and Nov. 5th, 1887.

² *Phil. Trans.* 1891, B, pp. 447, 449, 478 & 509.

³ *Loc. cit.*, p. 342.

⁴ *Loc. cit.*, p. 479.

such internuncial fibres.¹ "What initiates a motor process" they say,² "is to all intents and purposes motor." Or, as Ferrier³ puts it, "centres immediately concerned in effecting voluntary movements" are "as such motor." Both these

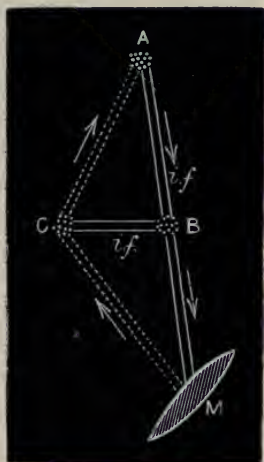


FIG. 1.

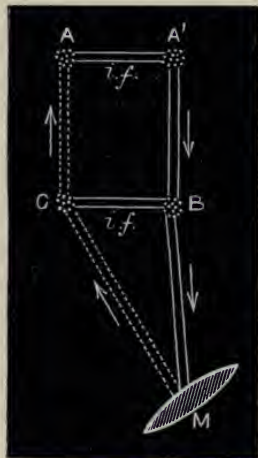


FIG. 2.

FIG. 1.—Diagram illustrating the origin of kinæsthetic impressions (so far as they come from muscle) and their relation to the production of voluntary movements, in accordance with my views.

A. Cerebral afferent (kinæsthetic) centre, receiving and registering ingoing impressions from muscle by way of an afferent spinal centre C; *i.f. i.f.*, two sets of internuncial fibres; B, spinal efferent or motor centre, which receives incitations from A, whence they are sent on to the muscle M.

∴ A is an afferent centre in the same sense that C is an afferent centre and each of them may initiate 'motor incitations' which pass along internuncial fibres to the motor centre B.

FIG. 2.—Diagram illustrating the origin of muscular sense impressions and their relation to the production of voluntary movements, in accordance with the views of Dr. Ferrier.

A. Cerebral afferent (tactile) centre receiving and registering ingoing impressions from muscle by way of an afferent spinal centre C; A' a supposed motor centre, which operates through commissural fibres upon the spinal motor centre B.

∴ No psychical processes are believed by either of us to be associated with the functional activity of the tracks represented by unbroken lines and the centres lying in their course.

I say that the functions attributed by Ferrier to A' are really performed by A as in Fig. 1, and that if A, A' existed as in Fig. 2, there ought to be two sets of excitable areas in each hemisphere.

¹ Since 1880 I have always restricted the term 'internuncial' to fibres which connect an afferent with an efferent centre; employing the term 'commissural' to those fibres that connect two afferent or two efferent centres with one another. (See "Brain as an Organ of Mind," p. 586).

² W. L. Mackenzie, in *BRAIN*, 1887, p. 433.

³ "Functions of the Brain," 2nd Ed., p. 348.

statements I believe to be altogether erroneous. As I have said elsewhere,¹ "The plan on which nervous centres generally are constructed, of whatsoever grade, makes it essential that the stimulus which awakens the activity of a 'motor' ganglion or centre shall come to it through connecting fibres from a 'sensory' ganglion centre, or knot of cells—that is, from cells which stand in immediate relation with ingoing fibres." Thus, we should not call a cortical centre for afferent impressions 'motor,' any more than we should call the group of ganglion cells on the afferent side of a spinal reflex arc 'motor.' In each case the nerve cells that receive the afferent impulses are in association with channels which convey 'motor incitations'; and in each case the stimulation of such internuncial fibres or of the centres from which they proceed would give birth to definite movements. The course of these internuncial fibres is for the most part horizontal in the spinal cord, though more rarely it may be an ascending one. But from the kinæsthetic centres in the brain the course of the internuncial fibres is downwards (in the pyramidal tract); hence the current is commonly spoken of, truly enough, as an "out-going current"; but with the effect, apparently, of fostering some confusion in the minds of not a few persons. It was apparently under the influence of some such confusion that W. L. Mackenzie,² a writer above quoted, penned the following sentences. If "we confine consciousness to sensory processes, then since sensory may excite motor processes, we must imagine consciousness suddenly ceasing on the nervous bridge—the internuncial fibres—between a sensory and a motor centre. If this be so the time has come for abolishing altogether the distinction of sensory and motor cortical centres; they are all sensori-motor." This seems to me to be an invalid conclusion, based upon a misconception as to the mode in which motor centres generally are stimulated. Certainly, there is no more reason why centres on the afferent side of the nervous system which happen to be situated in the cortex should be called 'sensori-motor,' than that such a term

¹ "The Brain as an Organ of Mind," p. 585.

² *Loc. cit.*, p. 432.

should be applied to similar afferent centres situated in the spinal cord. And yet it would be only upon such a basis that justification could be found for the oft-quoted view of Hughlings Jackson, cited by the same author (*loc. cit.*, p. 432) "that the physiological substratum of every mental process is a sensori-motor process."

From what has now been set forth, as well as from the facts and arguments detailed in a previous communication to this Society,¹ it seems to me quite clear that there is no reason for postulating the existence of cortical motor centres for the production of voluntary movements; that whatever the mode in which simple movements are produced, that is, whether they are voluntary or reflex, only one set of motor centres is called into play, and that these motor centres are situated in the bulb and in the spinal cord; and, further, that the functioning of motor centres generally is attended by no psychical accompaniments. In reference to this last point I have said elsewhere:²—"No ideal reproductions seem ever to take place in such centres; they are roused into activity by outgoing currents, and, so far as we have any evidence, the induction in them of molecular movements which, immediately afterwards, issue through cranial and spinal motor nerves to muscles are simply physical phenomena. These processes are apparently as free from subjective accompaniments as are the actual molecular processes thereby incited in the muscle itself." In fact the molecular motions that occur in motor centres "seem to lie even more truly outside the sphere of Mind than the molecular processes comprised in the actual contraction of a muscle; these latter are at least immediately followed by 'ingoing' impressions, whilst so far as we know—that is so far as any evidence exists—the former are not."

On the other hand, in common with James, and in part with Münsterberg and others, I maintain, that all the sensations resulting from movement are derived from ingoing

¹ "The 'Muscular Sense;' its Nature and Cortical Localisation," *BRAIN*, April, 1887.

² "The Brain as an Organ of Mind," p. 599. The lack of sensibility accompanying the action of motor centres has now been experimentally demonstrated by Gotch and Horsley (*loc. cit.* p. 510).

impressions; that such sensations are realized and revivable in idea in special sensory centres in the Rolandic area of the cortex, which are in intimate associational relation with visual, auditory, and other sensory centres; that the functional activity assumed to be performed by voluntary motor centres in the Rolandic area is really carried out by these sensory centres of kinæsthetic type, similarly located; so that in "the association of ideas" generally, as well as in the processes of Attention and Volition we have merely to do with the psychical activity of sensory centres and their annexes—and not of motor and sensory centres, with the former often dominating. Thus I hold that the process of Attention is in its essence sensory, though with inseparable motor accompaniments; while Volition, so far from "existing only in connection with the active organs, that is, with the muscular system,"¹ seems to represent merely some phases in the "association of ideas" and, as W. James puts it, is rather "a psychic or moral fact pure and simple, and is absolutely completed when the intention or consent is there"—he also maintaining that the supervention of motion upon its completion is a supernumerary phenomenon belonging to the department of physiology. The phenomena of Volition are, therefore, not the work of any special faculty or mysterious entity, nor are they carried on in motor centres; they are merely certain exemplifications of intellect in action; so that ample justification is found for the dictum of Spinoza, "*Voluntas et intellectus unum et idem sunt.*" Anything separate to be known as Will, is, in fact, a mere phantom—a kind of psychological ghost.

¹ Bain, "The Senses and the Intellect," 3rd. Ed., p. 559.

ON THE "INHIBITION" OF VOLUNTARY AND OF ELECTRICALLY EXCITED MUSCULAR CONTRACTION BY PERIPHERAL EXCITATION.

BY AUGUSTUS D. WALLER, M.D.

IN the course of experiments on fatigue I had occasion to repeat Fick's experiment¹ exhibiting an apparent inhibitory effect of direct tetanization upon voluntary contraction, inasmuch as direct strong induction shocks supervening upon an already existing strong voluntary contraction, do not add to but subtract from the latter.

Finding the lost time of the effect to be nearly one-tenth of a second (.09"), Fick attributes it to a central inhibition, whereas Mosso,² by a somewhat rough mode of estimation, finds it to be *in minimo* one-fifth of a second, and is of opinion that the phenomenon is one of direct inhibition at the periphery in the muscle itself, similar to the classical effect on the heart of vagus excitation, and to the relaxation of the crayfish claw or of the veratrinised frog's sartorius as described by Biedermann and others working with him.

I have been familiar with the phenomenon for some years, and attributed it in part to the direct excitation of antagonist muscles (a view which is alluded to by Fick, but only to be dismissed), in part to an ordinary reflex

¹ Fick. *Myographische Versuche am lebenden Menschen*, *Pflüger's Archiv.*, xli. p. 176. 1887.

² Mosso. *Ueber die Gesetze der Ermüdung*, *du Bois-Reymond's Archiv.*, 1890, p. 89.

remission of action, such as may be produced by any sudden strong sensation. More recently I have methodically examined the phenomenon in order to determine if possible whether it is of the simple mechanical character just alluded to, or whether it must be referred to a physiological inhibition — either a true inhibition in the periphery itself, as conjectured by Mosso, or a central action of arrest such as we are familiar with under many circumstances.

The method I followed was to take simultaneous tracings of the longitudinal and lateral alterations of the muscles of the forearm by a "dynamograph" grasped in the hand and a "bag-recorder" strapped to the forearm.¹ The dynamograph was a short stiff spring prolonged by a long light

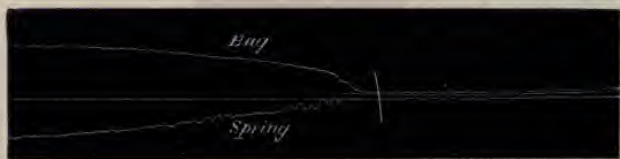


FIG. 1.

Simultaneous record of the longitudinal and lateral effects on the muscles of the forearm. The tracing shows the effect of strong faradisation upon strong voluntary contraction. Reading from right to left it commences with two lines indicating the level to which the two recording levers were raised by the voluntary contraction; at the point marked by a vertical arc faradisation was applied; the lateral tension of the entire muscular mass is increased, the longitudinal tension of the flexor muscles is diminished.

(The velocity of the recording surface, which is not marked, was about 45mm. per sec., *i.e.*, as in fig. 2.)

lever, so that the flexor muscles of the forearm contracted under approximately isometric conditions. The bag-recorder needs no special description; it was identical with that used by Dr. de Watteville and myself in experiments on electrotonic alterations of excitability.

A simultaneous record of Fick's experiment with the two

¹ Cf. BRAIN, 1891, pp. 204-206.

instruments is, at the very first glance, suggestive of the simple mechanical explanation by antagonist contraction; the record of the longitudinal tension frequently exhibits a fall, while that of the lateral tension exhibits a rise, and if the records be taken on a surface at a suitable speed, the lost time is sometimes as short as that of a direct effect. The obvious explanation, as it seems to me, is that when the hand is grasped to the full the flexors are in maximal, the extensors in sub-maximal action, and that when the whole muscular mass is faradised, the increased rigor, chiefly of the extensors, adds to the lateral, and subtracts from the longitudinal tension.

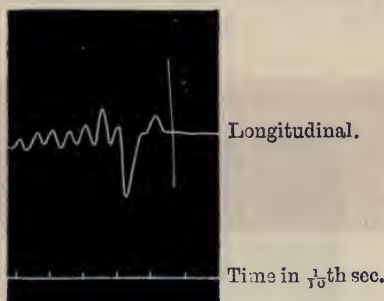


FIG. 2.

Diminution of voluntary abduction of index finger, caused by a single break induction shock, applied to the abductor indices. The recording spring is deflected by the index finger, with the hand disposed as figured by Fick (*Pflüger's Archiv.*, vol. xlv., p. 300); the moment of excitation is indicated by the vertical arc: the first effect is a slight rise of abduction, followed by a more pronounced fall of abduction, commencing about $\frac{1}{10}$ sec. after excitation; (the after oscillations are caused by the spring and lever, which was unduly long and flexible); time is marked in $\frac{1}{10}$ th sec.

The conditions of experiment were, however, not identical with Fick's,¹ and it was obviously necessary, before proceeding further, to repeat his experiment as exactly as

¹ Fick does not state whether he used a bipolar or a unipolar mode of excitation; I have therefore used both. It appears from the context that he obtained the negative effect with single induction shocks; I have used both single shocks and faradization, according to unipolar and bipolar application.

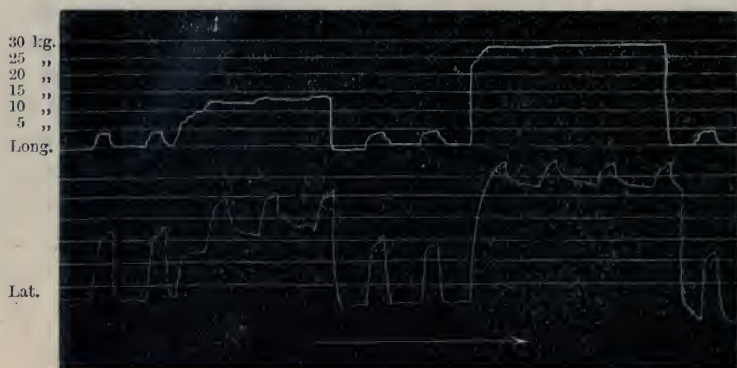


FIG. 3.

Tetani by direct faradisation (3000) alone, and superposed upon submaximal voluntary tetani (12 and 28 kg.). Simultaneous longitudinal and lateral records. Left forearm. (Tracing reduced by photography.)

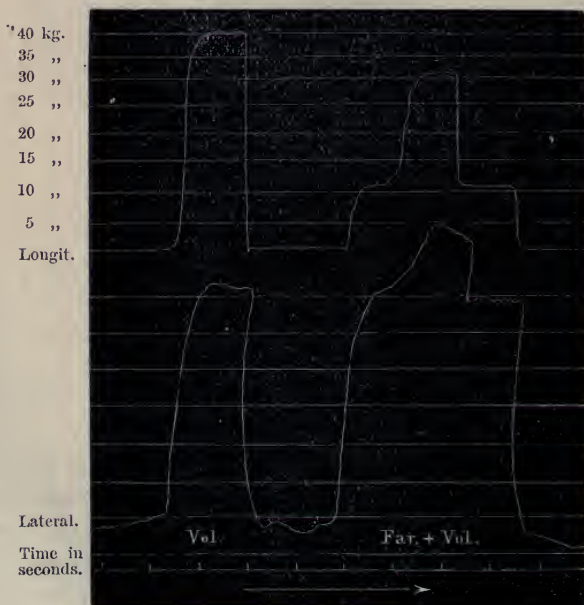


FIG. 4.

Maximal voluntary effects, alone and superposed upon direct tetanus by strong faradisation (6000). Simultaneous longitudinal and lateral records. Left forearm.

possible, if only to see whether his time datum could be verified or accounted for. I therefore repeated the experiment with a fine electrode on the abductor indicis under isometric conditions. Unlike Fick, I found that the strength of faradisation required to produce the effect was far too great to allow the assumption that antagonist muscles were not excited. With the currents employed, and supporting the hand as figured by Fick, a true reflex start with a time-interval of about one-tenth of a second was frequently obtained, exhibiting a less action of the abductor just as described by Fick, but not, in my opinion, admissible as a specific interference effect. Indeed, the reflex remission occurred as often after the end as after the beginning of the period of faradisation. I was therefore obliged to conclude that the effect is a simple antagonist effect, or an ordinary reflex start, and that the experiment given by Fick is not an illustration of inhibition in the strict physiological acceptation of the term.

I then returned to observations on the forearm muscles where records of lateral and of longitudinal effects could be combined. To get a general view of the mutual effects upon each other of direct and of voluntary contractions, I took series of direct excitations of various strengths superposed upon voluntary tetani of various strengths, and maximal voluntary tetani superposed upon direct tetanisation also of various strengths. The results are embodied in the tables below, and figs. 3 and 4 give examples of the mode of record. It will be understood that the numbers expressing the longitudinal tension are in kilogrammes as read by the effects on the graduated spring, while those of the lateral effect express no unit, but simply the height of lever excursions caused by the swelling of the contracting forearm. In all cases, however, the two indications are simultaneously taken. It will also, no doubt, be noticed that the longitudinal effects of direct are very feeble in comparison with those of voluntary contraction; this is always the case, and has already been commented upon by Fick.

LEFT ARM.—*Maximum voluntary contraction plus direct muscular faradisation of various strengths.*

		Longitudinal Tension.	Lateral Tension.
—————	Farad. 2000	0 Kilos.	10 mm.
Vol. Contr. + , ,	, ,	45+0 , ,	23+1 , ,
—————	Farad. 3000	5 , ,	20 , ,
Vol. Contr. + , ,	, ,	42+0 , ,	26+3 , ,
—————	Farad. 4000	7 , ,	25 , ,
Vol. Contr. + , ,	, ,	42+0 , ,	25+5 , ,
—————	Farad. 5000	8 , ,	25 , ,
Vol. Contr. + , ,	, ,	41+0 , ,	25+5 , ,
—————	Farad. 6000	10 , ,	30 , ,
Vol. Contr. + , ,	, ,	47—5 , ,	25+5 , ,
—————	Farad. 7000	10 , ,	30 , ,
Vol. Contr. + , ,	, ,	43—3 , ,	22+5 , ,
—————	Farad. 8000	10 , ,	27 , ,
Vol. Contr. + , ,	, ,	43—5 , ,	22+5 , ,
—————	Farad. 9000	12 , ,	26 , ,
Vol. Contr. + , ,	, ,	40—5 , ,	20+5 , ,

We learn from these observations—

(1) That the longitudinal tension of the flexors in the forearm increased to its maximum by voluntary action cannot be further increased by direct muscular faradisation.

(2) That with very strong direct faradisation, supervening upon maximum voluntary contraction, the longitudinal tension undergoes a diminution.

(3) That the lateral tension of the muscles in the forearm, increased to its maximum by voluntary action, can be further increased by direct muscular faradisation.

(4) That an increased lateral tension and a diminished longitudinal tension may be synchronous events.

The synchronous diminution of longitudinal tension and increase of lateral tension is probably due to increased contraction of extensor muscles. This, however, is not yet proved; it is a conceivable alternative that the supervention of direct faradisation upon voluntary tetanus should cause a lateral increase and a longitudinal diminution of tension.

RIGHT ARM.—*Direct muscular faradisation of various strengths plus maximum voluntary contraction—i.e., voluntary superposed on faradic contraction (see also fig 4).*

		Longitudinal Tension.	Lateral Tension.
—————	Vol. Contr.	45 Kilos.*	30 mm.
Farad. 2000 +	„	7+37 „	10+20 „
—————	Vol. Contr.	42 „	30 „
Farad. 2500 +	„	6+35 „	20+10 „
—————	Vol. Contr.	44 „	30 „
Farad. 3000 +	„	7+29 „	25+ 8 „
—————	Vol. Contr.	45 „	29 „
Farad. 3500 +	„	9+26 „	27+ 8 „
—————	Vol. Contr.	45 „	31 „
Farad. 4000 +	„	9+25 „	27+10 „
—————	Vol. Contr.	42 „	29 „
Farad. 4500 +	„	11+23 „	30+10 „
—————	Vol. Contr.	42 „	30 „
Farad. 5000 +	„	10+20 „	30+10 „
—————	Vol. Contr.	42 „	30 „
Farad. 6000 +	„	11+20 „	30+10 „
—————	Vol. Contr.	41 „	30 „
Farad. 7000 +	„	11+20 „	30+10 „
—————	Vol. Contr.	42 „	30 „
Farad. 8000 +	„	12+18 „	30+10 „

We learn from these observations—

(1) That a maximal longitudinal tension by direct faradisation can be further increased by voluntary action.

(2) That the stronger the direct faradisation the smaller the maximum voluntary increment.

(3) That the longitudinal effect of faradisation, *plus* voluntary action, is smaller than that of voluntary action alone.

(4) That a maximum lateral tension, by direct faradisation, can be further increased by voluntary action.

(5) That the maximum lateral tension by faradisation, *plus* voluntary action is greater than the maximum lateral

tension obtainable by voluntary action alone or by faradisation alone.

These differences of effect are very probably due to the extensor group of muscles. But, again, we must recognise that the reality of this factor is not proved, and that it remains possible to imagine a maximum lateral tension in a given muscle caused by faradisation to be further increased by voluntary action. But, on general review, it is clearly more probable that the varying relations between longitudinal and lateral effects are mainly due to the extensor element.

LEFT ARM.—*Submaximal voluntary contraction of various strengths plus direct muscular faradisation of constant strength—i.e., faradic superposed on voluntary contractions.*

Volunt. Contr.	Faradn.	Longitudinal Tension.		Lateral Tension.
None	4000 Units	6	Kilos.	22 mm.
10 Kilos. +	" "	+2	"	+10 "
None	" "	6	"	24 "
12 Kilos. +	" "	+1.5	"	+ 8 "
None	" "	5	"	20 "
12 Kilos. +	" "	+1.5	"	+ 7.5 "
None	" "	5	"	17 "
5 Kilos. +	" "	+3	"	+12 "
None	" "	4	"	17 "
7 Kilos. +	" "	+2	"	+ 8 "
5 " +	" "	+2	"	+10 "
11 " +	" "	+1.5	"	+ 8 "
None	" "	4	"	18 "
None	3000 Units	5	"	15 "
20 Kilos. +	" "	+0	"	+ 8 "
None	" "	4.5	"	13 "
27 Kilos. +	" "	+0	"	+ 5 "
None	" "	4	"	12 "
40 Kilos. +	" "	+0	"	+ 3 "

0 Kilos.	3000 Units	4 Kilos.	15 mm.
5 "	+ " "	+1.5 "	+ 8 "
0 "	" "	4 "	15 "
6 "	+ " "	+1.5 "	+ 7.5 "
0 "	" "	4 "	13 "
3 "	+ " "	+2.5 "	+ 8 "
0 "	4000 "	6.5 "	17 "
11 "	+ " "	+2 "	+ 8 "
0 "	" "	6 "	17 "
6 "	+ " "	+2.5 "	+10 "
0 "	" "	5.5 "	17 "
4 "	+ " "	+3 "	+10 "
12 "	" "	+1 "	+ 5 "
0 "	+ " "	5 "	13 "
20 "	+ " "	+0 "	+ 3 "
0 "	" "	5 "	13 "
4 "	+ " "	+1.5 "	+ 8 "

[NOTE.—Each group of numbers separated by horizontal lines refers to a series of observations taken on a given day. The numbers in the last two columns give only the effects of faradisation alone, or superposed upon voluntary contraction.]

LEFT ARM.—*Single break induction shocks of various strengths superposed upon maximum voluntary contraction of between 40 and 30 kilos. longitudinal tension. Only the lateral effects are given.*

	Before.	During Vol. Contr.	After.
Farad. 2000	2.5 mm.	30+ 2 mm.	3 mm.
" 3000	4 "	30+ 3 "	5 "
" 4000	10 "	35+ 5 "	12 "
" 5000	15 "	35+ 6 "	18 "
" 6000	20 "	35+ 7 "	23 "
" 7000	25 "	35+ 7 "	25 "
" 8000	30 "	30+10 "	30 "
" 9000	32 "	30+12 "	34 "
" 10000	30 "	35+12 "	30 "
" 11000	35 "	35+13 "	35 "
" 12000	35 "	35+13 "	35 "

[NOTE.—The numbers of longitudinal tension are not worth giving, the effects being trifling or variable; it is better to give cuttings of two groups, the second of which shows the negative isometric effect which has been commented upon by Fick, and which is the main motive of this paper.]

Single break induction shocks of constant strength superposed upon submaximal voluntary contraction.

VOL. CONTR.		FARAD.	Longit. T.	Lateral T.
0 Kilos.	6000 Units		2.5 Kilos.	15 mm.
6 "	+ 6000 "		6+1.5 "	17+ 8 "
0 "	6000 "		3 "	16 "
10 "	+ 6000 "		10+2 "	20+10 "
0 "	6000 "		3.25 "	20 "
4 "	+ 6000 "		4+3 "	7+15 "
0 "	6000 "		3 "	22 "
10 "	+ 6000 "		10+1.5 "	18+12 "
0 "	6000 "		3 "	20 "
0 "	5000 "		2 "	15 "
6 "	+ 5000 "		6+1.5 "	17+ 8 "
9 "	+ 5000 "		9+2 "	20+10 "
0 "	5000 "		3.25 "	20 "
4 "	+ 5000 "		4+3 "	10+15 "
0 "	5000 "		3.5 "	25 "
9 "	+ 5000 "		9+1.5 "	20+12 "

[NOTE.—These tables of numbers give, however, a very imperfect idea of the actual alterations; the irregular cases which, in a summary view, are obviously exceptional, assume undue prominence when put into series of numbers. Here, again, it is better to give cuttings of one or two short groups.]

From the foregoing data and comments it appears that in the combined effects of voluntary and faradic contraction there is no cogent nor even plausible evidence of true inhibition; the negative longitudinal effect with positive lateral

effect, illustrated in figs. 1, 5 and 7 is evidently due to the supervening action of antagonist muscles—*i.e.*, Munk's "antagonistische Hemmung"; this antagonist action can be produced by direct excitation, or it may occur as a reflex.

The facts (*a*) that a maximum voluntary longitudinal effect cannot be increased by direct excitation, (*b*) that a maximum longitudinal effect by direct excitation can be in-

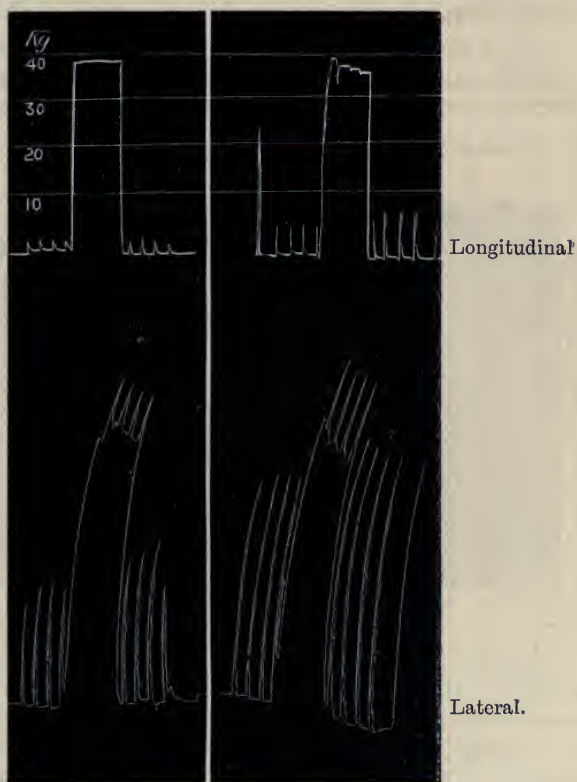


FIG. 5.

Contractions caused by single induction shocks (5000 and 10000) alone and superposed upon maximum voluntary tetanus. Simultaneous longitudinal and lateral records. In the longitudinal record of the second group, the negative effect described by Fick is visible; it is accompanied by a positive effect seen in the lateral record. Left forearm.

creased by voluntary action contain no proof of a physiological interference between the two forms of excitation. It is obvious that if in muscles of given total power, a portion

of energy be called forth by one kind of excitation, a smaller portion than the total remains that can be elicited by the other kind of excitation. We have seen that greater energy can be elicited by voluntary than by electrical excitation, and it is therefore reasonable to find that the effect of the former can add to the effect of the latter, and not *vice versâ*. The difference is, no doubt, mainly due to an intrinsic superiority of the natural as compared with the artificial mode of excitation, but it is probably also in part a consequence of the fact that by strong direct excitation antagonist muscles are brought into excessive action as compared with their action in strong voluntary motion.

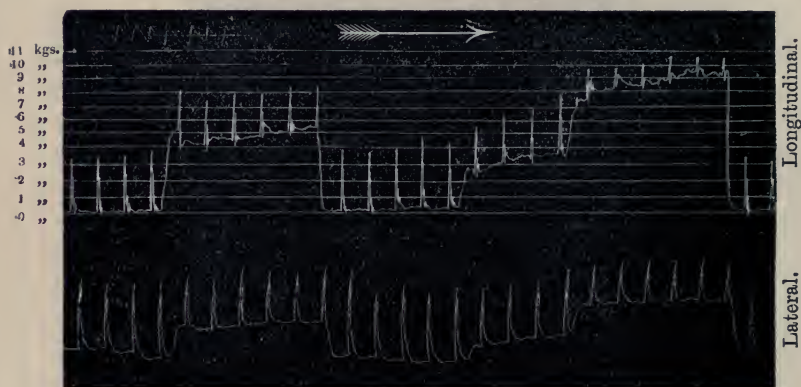


FIG. 6.

Contractions by single induction shocks (6000) alone and superposed upon submaximal voluntary tetanus (4 to 5 and 8 to 9 kilos). Simultaneous longitudinal and lateral record. Left forearm. (Tracing reduced by photography.)

The only remaining datum which apparently favours the notion of an interference between the natural and the artificial effect is that illustrated in fig. 4—viz., the longitudinal effect of direct faradisation *plus* voluntary action is smaller than that of voluntary action alone, and the stronger the faradisation the smaller the maximum voluntary increment. I think that the effect is to be explained as follows:—(a) It is in part due to the action of antagonist muscles; (b) it is in part due to the fact that the greater the portion of total

energy called up by faradisation, the smaller the remaining portion to be called up by voluntary action. These two points have been considered, but a third possibility remains which leads us very close to the notion of inhibitory interference in muscle; (c) it may be due to the fact that the dislocation of molecules in muscle caused by electrical excitation is different from the dislocation caused by voluntary excitation, and that the former interferes with the latter. This is a fundamental query at the very root of the main question we are dealing with, which demands searching analysis, verbal and experimental; but I can venture to say at present little beyond what has already been said in a previous paper.¹

There is some very deep-seated difference between maximum direct contraction and maximum voluntary contraction; the former is ineffectual and injurious as compared with the latter, which is powerful and harmless; and that the molecular dislocation is not identical in the two modes of contraction is indicated by the fact that, with a given longitudinal effect in the two cases, a greater lateral effect is associated in faradic than in voluntary tetanus. It is reasonable to suppose that a given molecular dislocation by faradisation yielding a comparatively small dynamic effect subtracts from the residual dislocation possible by voluntary action in such degree that the dynamic effect of that superadded voluntary action is much less than normal. But even if this be a correct interpretation, does the effect amount to a true interference phenomenon, an inhibition of voluntary by electrical excitation? I think not; for we do not have any arrest of previous voluntary by subsequent electrical contraction, we have only a diminution of subsequent possible voluntary by previous electrical contraction. To sum up—given the existence of antagonist muscles and of current diffusion, we have a simple explanation of the minus dynamic effects above described; but we may not assume that this explanation fully covers the phenomena to the exclusion of other factors. We have reason to admit that in voluntary added to faradisation contraction, the effect is in part due to

¹ BRAIN, 1890, p. 198.

a wasteful usurpation by the latter (faradisation) of contractile material which would otherwise be at the disposal of the former (voluntary action) which is more economical as regards the dynamic effect associated with a given amount of molecular dislocation. We have no right to assert that this is an instance of true physiological inhibition. But a denial of the existence of such a phenomenon is not justified by the data—the case stands thus :—we may deny that the assertion is proved by the evidence, we may not assert that the phenomenon does not exist.

Casting about for some instance in the body in which it may be possible to directly excite a muscle without remaining under the suspicion that antagonist muscles are simultaneously excited, it occurred to me to try the elevators of the lower jaw.¹ In the case either of the temporal or of the masseter it is easy to take an isometric indication of the longitudinal effect as well as a record of the lateral effect, and to examine the influence of direct faradisation superposed upon maximum voluntary contraction.

To record the bite I used a kind of gag, the upper half of which was fixed and served as a "point d'appui" to the upper jaw, while the lower half was in rigid connection with a very stiff spring the minute deflection of which was recorded as already described.

I never obtained the slightest sign of let-go of the voluntarily contracted temporal or masseter muscles on the superposition of faradisation as strong as could be borne. And the record of the lateral effect taken by means of a bag strapped over the muscle, or inserted in the mouth between the gums and cheek, exhibited no augmentation or diminution of rigidity coincident with the electrical stimulation. The unpleasant effects of electrical currents applied to the sides of the head, prevented me from examining these muscles in a satisfactory manner, as regards the superposition of voluntary upon faradic contraction; I could always increase the latter by the former, but the currents

¹ These muscles have been previously employed by Gad for a different purpose, but for the same reason—viz., their independence of antagonist muscles. Cf. *Orschansky. du Bois-Reymond's Archiv.*, 1889, p. 173.

were only of moderate strength, and I took no exact measurements of the varying effects with varying strengths.

I also made the following experiment, which although it *proves* little, nevertheless, imitates and exhibits, under known conditions, the effect of increased antagonist effect, supervening upon the contraction of a group of muscles, and in less degree of the antagonists themselves.

Two exciting electrodes were fixed to the arm, so as to excite, respectively, the median and the musculo-spiral nerves. The muscular contractions were simultaneously recorded by spring and by bag. During tetanisation of the median nerve

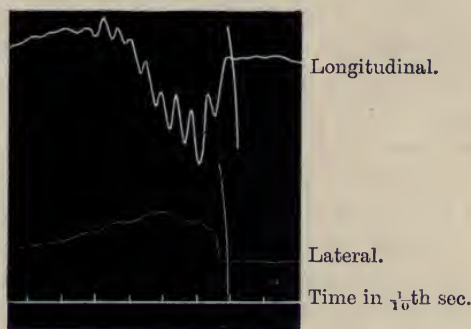


FIG. 7.

Effect of tetanisation of musculo-spiral nerve, superposed upon tetanisation of the median nerve. Simultaneous longitudinal and lateral record; the effect is negative in the former, positive in the latter. The oscillations are due to the long flexible lever.

(putting into contraction chiefly flexor muscles of the forearm), faradisation from a second coil is applied to the musculo-spiral (putting into contraction chiefly extensor muscles of the forearm). The effects on the longitudinal and lateral tensions are precisely those which we saw when direct excitation was superposed upon voluntary flexor contraction, *i.e.*, longitudinal tension is diminished, lateral tension is increased.

The interest attaching to the presence, or absence, of true peripheral inhibition in muscle itself, is closely connected with a question which must often present itself to the physiological neurologist. What happens when we cease

a voluntary action? Does an anti-motor mandate pass along an efferent nerve and interfere with the muscular effect of a pre-existing motor mandate? Or, does a motor mandate simply cease to pass with the cessation or arrest of volition. In other words, is the arrest of motor action consummated in the nerve-centre, or in the muscle?

So long as positive proof of peripheral inhibition is not forthcoming, we must choose as the more probable answer, that arrest of action, like the commencement of action, is of central origin.

The mechanical arrest of action by the anti-effect of antagonist muscles — Munk's antagonistische Hemmung (*du Bois-Reymond's Archiv.*, 1881, p. 553)—contributes, of course, to the effect in normal movements. But, that apart from this purely mechanical positive antagonism, there is a negative action of the motor muscles when a movement ceases, and that this negative action is the main effect, is shewn by the simultaneous record of the lateral effect. If in the commencement of release of the deflected spring, the antagonistic muscles play chief part, there is a prolongation of the lateral, as compared with the longitudinal indication, similar to that which I have shewn to occur in fatigue (*BRAIN*, 1890, pp. 208, 211, figs. 11 and 13), but under ordinary conditions there is no such prolongation of the lateral effect by the muscles of the forearm at the end of a flexor effort of the fingers, and we may conclude from this that contraction of extensors does not play the chief part in the cessation of flexion.

Experiments directed to this question as to the existence of anti-motor nerve-impulses to voluntary muscle, have already been made by Orschansky, under Gad's direction,¹ with entirely negative results. The masseter was the object of observation, and the point of comparison was the reaction-time of its "*positive*" response by contraction, and of its "*negative*" response by relaxation. No sensible difference was found to exist between the two times.

I addressed myself to the question in a somewhat

¹ Orschansky. "Wilkürliche Impulse und Hemmungen," *du Bois-Reymond's Archiv.*, 1889.

different manner. At first—and fully alive to the possible complication by antagonist action—I used the muscles of the two forearms; taking a simultaneous record of the longitudinal and lateral effects of both, I examined the lost times of the various records obtained by an act of volition, by which contraction was simultaneously begun on one side, and ended on the other side.

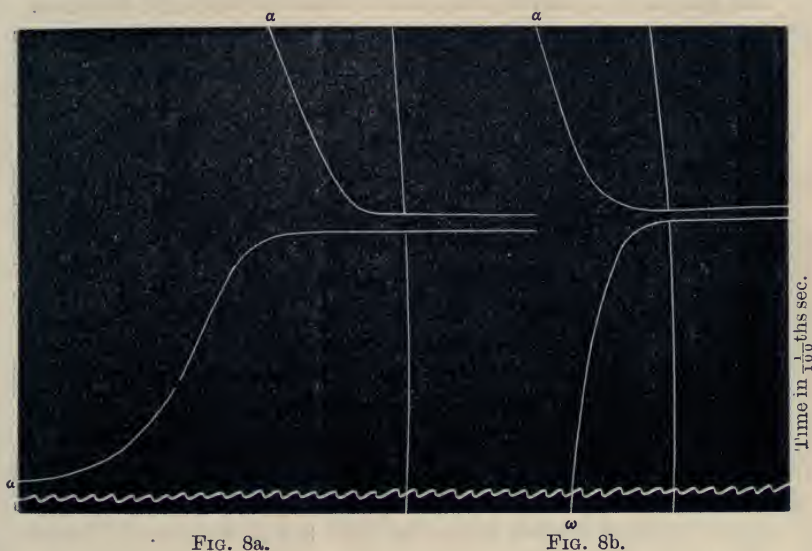


FIG. 8a.

FIG. 8b.

8a. Simultaneous positive and negative contraction of the two hands by faradisation of the flexor muscles. The upper line α is that of the flexor grasp, caused by faradisation; the lower line ω is that of the flexor cessation of grasp, caused by the cessation of faradisation; the corresponding arcs on the two lines indicate, respectively, the commencement of faradisation on one side, and its cessation on the other.

8b. Simultaneous positive and negative voluntary grasps of the two hands. The vertical arcs indicate correspondence of levers. Both these tracings are longitudinal records taken by a double dynamograph.

The attitude of attention was naturally found to be of some influence on the result. Thinking to the left was apt to cause the left side to act, or cease to act, a little before the right, and *vice versâ*. Thinking in the middle line, *i.e.*, concentrating the attention upon an object

straight ahead, and working the two sides as automatically as possible, the commencement of action on one side, and the cessation of action on the other, were as nearly as possible, simultaneous.

There is, at first sight, nothing surprising in this result; if we keep free of the knowledge we possess of the physiology of muscular contraction, we may take as matter of course, that in the successive co-ordination of complicated manœuvres, simultaneously willed positive and negative effects should begin simultaneously.

It is only when a little more reflection is given to the matter, that its interest becomes apparent.

If a muscular tetanus, by direct excitation, be recorded, and the time of commencement and of termination of the faradisation be indicated in the usual manner, it will be found that the effect begins a little later than the cause (latent period of contraction), and that the effect considerably outlasts the cause (period of maintenance of contraction). If we, simultaneously excite one muscle and cease to excite another, using direct faradisation, we shall find that the second muscle continues contracted for a short period after the first muscle has commenced to contract.

Now, nothing of this kind happens in voluntary action and cessation of action. The initial points of both events simultaneously willed are practically simultaneous.

And we are left in presence of two alternative suppositions, in accordance with either of which the phenomenon may be explainable, but between which I can, at present, see no experimental mode of probation—(a) the origin of one impulse, and the end of another impulse, occur simultaneously in the cortex cerebri, but the negative impulse gets to the muscle sooner than the positive impulse, or (b) a single volition of two phenomena, one positive, one negative, does not produce the two events simultaneously but successively, and at that interval which the organisation of past experience has established as the interval at which a positive and negative act must occur in the cerebrum, if they are to produce their effect simultaneously at the periphery. The co-ordination in time must be such that they are asynchronous in the

centre to be synchronous at the periphery. Although we cannot experimentally decide between these two alternatives, the second one is, obviously, by far the more probable of the two.

We have, so far, failed to find any cogent evidence on human muscle, of a true interference phenomenon between voluntary contraction and electrical excitation by induced currents. The smaller effect of voluntary contraction in the presence of faradic contraction, and the smaller effect of direct excitation in the presence of voluntary contraction, do not contain any cogent proof of an interference effect, but are intelligible enough as the effects of the fact, that if some energy is elicited by one agency, less energy can be elicited by the other, supervening upon the first.

There remain to be examined the effects of the galvanic current upon voluntary contraction; and I undertook this portion of the inquiry with some expectation of an affirmative result, in view of the theories and experiments of the Prague School, more especially the relaxant effect upon the adductor muscle of the claw of the cray-fish, caused by excitation of nerve, and the anodic closure inhibition of non-electrical tonic contraction, as investigated by Biedermann. In doing this it was, of course, necessary to bear in mind, that it is not possible on the human subject to obtain pure polar effects; but that we may, at most, hope to obtain a polar anodic effect, predominating over a peripolar kathodic effect. The questions accessible to experiment under this head are:—

1. Can the voluntary impulse be anodically blocked?
2. Can voluntary contraction be anodically inhibited?
3. Can an electrical excitation be anodically blocked?
4. Can an electrical contraction be anodically inhibited?

The related question as to the effects of the galvanic current upon faradic contractions was already familiar to me, from a systematic investigation of the subject on man, which I made ten years ago, with my friend Dr. de Watteville.¹

I may say at once, that to questions (1) and (2), the ex-

¹ Waller and de Watteville. "On the influence of the Galvanic Current upon the Excitability of the motor nerves of Man," *Phil. Trans.*, Royal Soc., 1882.

perimental answer proved to be negative; whereas to (3) and (4) it is affirmative, but only in the sense of Pflüger's classical investigations (1859), showing that the anode is depressant of excitability. In fact, the experiments made to test questions (3) and (4) became almost identical with those by which Pflüger's results have been verified as covering the case of human nerve.

Considered seriatim :—

(1) To examine whether the voluntary impulse can be anodically (or kathodically) blocked, I made a series of maximum voluntary flexor efforts, with periods of maximum endurable anodic (and kathodic) polarisation of the median nerve for about $\frac{1}{4}$ min., at intervals of about one min. or less. No effect whatever was produced during the flow of the galvanic current, and the fatigue decline proceeded as if no galvanic current were present.

(2) To examine whether the voluntary contraction can be anodically (or kathodically) inhibited, I repeated the experiments with the anode (or kathode) applied to the flexor muscles. No effect was produced.

(3 and 4) To examine whether an electrical excitation (by the induced current) can be blocked or inhibited by a galvanic current, I employed two pairs of electrodes connected with a battery and coil respectively. Each of the two exploratory electrodes was applied to the left forearm over the flexor muscles and the median nerve respectively; each of the two indifferent electrodes was fastened to one leg, and it is obvious that, with this disposition, there was a variable coincidence in the distribution of the two currents, and consequent superposition of the induced upon the galvanic current. The effects corresponded with the conditions, the induced currents produced smaller or larger contractions (isometrically recorded) according as the points of excitation were brought under anodic or kathodic influence; and this was the case whether the exploring nerve-electrode was connected with the coil and the exploring muscle electrode with the battery, or *vice versa*.

Seeing that, with two pairs of electrodes, the two circuits must be more or less mutually derivative, I then used only

a single pair of electrodes, placing the secondary coil in the battery circuit, and applying the break induced current with and without the battery circuit, with the single exploring electrode applied to the median nerve or to the flexor muscles. This disposition of circuit was, in fact, identical with that which Dr. de Watteville and I selected for the investigation of polar alterations of nerve-excitability caused by the galvanic current, and the result presented no feature of novelty—*i.e.*, the effect of cathodic excitation, whether

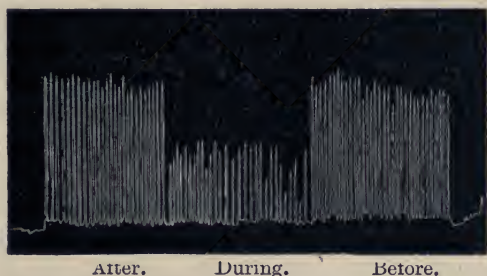


FIG. 9.

Dynamographic tracing of the effects of single induction shocks (kathodic break 7000) before, during, and after the passage of a constant current (anode, 30 cells). Exploring electrode applied to the flexor muscles of the left forearm.

polar or peripolar, was increased during cathodic polarisation, diminished during anodic polarisation. It is, however, worth mentioning that, whereas in the 1882 results, the method adopted left undetermined whether an increased or diminished record was due to increased or diminished contraction of a given amount of muscle, or to the contraction of a larger or smaller amount of muscle, the method used in the present inquiry unmistakably exhibits the greater or smaller contraction of a given group of flexor muscles. And I may add that, while the 1882 inquiry was confined to the investigation of the excitability of nerve, I have taken the present opportunity of verifying that the phenomena of increase and diminution are precisely similar when excitation is directly applied to muscle—*i.e.*, to muscle *cum* intra-muscular nerve.

It is a question of words whether or no we choose to apply the term "inhibitory" to this anodic effect upon excitability or excitation. There is an obvious resemblance between the phenomenon of arrest and that of anodic depression which invites the word; a regular series of muscular systola produced by induction shocks is suddenly diminished or suspended by anodic influence, or a muscular tetanus is cut down during anodic influence, just as the heart's systola are diminished or suspended by vagus influence; or, as in Biedermann's experiment, a local contraction of veratrinised muscle is partially resolved during anodic influence. And, although the connotations of the term "inhibitory" to which we have become accustomed in the case of the heart and of nerve centres, are very different from those which were formerly attached to the term "anelectrotonic," I do not think that our present state of knowledge justifies either an affirmation or a denial of essential similarity of mechanism between these two phenomena of arrest. The symmetry of view presented by Hering's exposition¹ of positive and negative trophic changes in the retino-cerebral apparatus, and of the relation between similar changes, and anodic and kathodic effects and after-effects which take place in nerve and in muscle, is most seductive, but we must remember the highly

¹ Hering. *Zur Lehre vom Lichtsinne*. Wien., 1874.—*Zur Theorie der Vorgänge in der lebendigen Substanz*, "Lotos," vol. ix., Prag., 1888.

I have made some experiments on the electrical effects of different parts of the spectrum acting upon the frog's retina, to see whether any objective differences could be demonstrated in any correspondence with Hering's "assimilatory" and "dissimilatory" groups of colours. I obtained no results pointing in this direction, but found (as had been previously found by Dewar and M'Kendrick) that all the colours act in the same sense upon the retina. I do not, however, place any reliance on the electrical test either in support of, or in opposition to, Hering's theory of antagonistic colours. We have very little positive knowledge of a relation between electrical change and trophic analysis or synthesis, apart from visible mechanical change; even if we might admit as established an association between analytic change and electrical negativity, and between synthetic change and electrical positivity, we might explain the failure to observe opposite effects with yellow v. blue and red v. green, as being due to the whiteness (weisse Valenz) of these colours, or to their associated heating effects. And although we may cut off the invisible and most heating rays of the spectrum by an alum cell, radiant energy will still pass through the cell, which is not exclusively "luminous" but also "heating"; we may not in fact say that the electrical change of the retina is an effect of light or of heat, but only that it is an effect of the radiant energy manifested as light and heat. Dewar and M'Kendrick (*Trans. R.S., Edin., 1873, p. 141*) found that light passed through solutions of various colours acted in the following order of strength: yellow, green, red, blue.

hypothetical character of that view, while recognising the hypothesis itself as an intellectual framework into which many fundamental phenomena of living matter fit themselves with most suggestive regularity. Greater integration is associated with rest, greater disintegration is associated with action, but whether the trophic movement is the cause of the functional expression in either case or in both, or *vice versa*, is still an open question.

A priori, it is probable that action is the consequence of greater disintegration, and that greater integration is the consequence of rest. It is less probable that disintegration is the consequence of action and that rest is the consequence of integration; or, shortly expressed—muscle discharges to act, and stops to recharge.

A posteriori there is very little experimental indication to show which of these probabilities must be adopted, as the nearer to the actualities of sequence between nutrition and action. We have, as yet, no sufficient warrant for going beyond the classical doctrine of the French School of Physiology, as expounded by Claude Bernard, into either of the trains of thought which are pleaded for—in Germany by Hering, in England by Gaskell—the anodic assimilatory inhibition of the former, or the anabolic inhibition of the latter.

We are passing up beyond the ground level of positive phenomena into the regions of hypothesis and speculation. To do so is unavoidable, and even desirable, provided that we clearly recognise and distinguish between scientific imagination, and scientific observation. The particular question of efferent inhibitory nerves to voluntary muscle, the broader question of assimilatory and dissimilatory stimuli of visual nerve matter, and of all living matter; the offshoots of that question which have sprung up in recent years, anodic inhibition (Biedermann), anabolic inhibition (Gaskell), are hypotheses actually under examination; things are rarely seen until looked for, imagination precedes and guides observation, and the doctrinal tendencies adverted to above are leading the researches of many workers in Germany, in Italy, and in England. And failing careful sifting, we are

apt to confuse things imagined with things seen, or to place excessive doctrinal importance upon unimportant and insufficient facts.

Reviewing the position as to theory and as to facts, I think that as regards the former, it is unsafe to go much beyond the teaching of Claude Bernard. The position, in so far as it is legitimised by experimental data, and beyond which, I think, the ground becomes uncertain and treacherous, is most elegantly, as well as most emphatically and cautiously expressed in Bernard's *Phénomènes de la Vie*, Paris, 1879, Vol. II., and the passage deserves to be quoted *in extenso*.

On peut distinguer deux ordres de phénomènes :

1° Les phénomènes de fonctionnement, ou encore d'usure ou de *destruction vitale* ;

2° Les phénomènes de formation, ou de création vitale, ou encore de *synthèse organique*.

Cette systématisation, à laquelle j'ai été conduit par un examen approfondi, m'a paru la plus conforme à la réelle nature des choses, à la fois compréhensive et féconde : elle se fonde uniquement sur les propriétés universelles de l'élément vivant, abstraction faite des moules spécifiques dans lesquels la substance vivante est engagée.

Les deux types ne sont jamais isolés : ils sont indissolublement connexes, et la vie de quelque être que ce soit est caractérisée précisément par leur réunion et leur enchaînement : ils représentent les deux phases du travail vital.

Cette vérité constitue, ainsi que nous l'avons dit ailleurs, *l'axiome de la physiologie générale*. On peut être assuré que toute doctrine qui serait directement ou indirectement en contradiction avec cette donnée fondamentale est fausse, et que le principe de l'erreur est précisément dans cette contradiction.

Les phénomènes de *destruction organique* ont pour expression même les manifestations vitales. *Toute manifestation vitale est nécessairement liée à une destruction organique*.

Les phénomènes plastiques ou de *synthèse* régénèrent les tissus, réparent les pertes, rassemblent les matériaux qui devront être dépensés de nouveau. C'est un travail intérieur, silencieux, caché, sans expression phénoménale évidente, travail d'une nature plus spéciale, plus vitale en quelque sorte, car il n'a pas d'analogue en dehors des organismes.

Il importe de ne pas perdre de vue les deux phases du travail physiologique, l'organisation et la destruction fonctionnelle. Elles se distinguent de toutes les façons : par leur expression phénoménale, par leur nature chimique, par leurs conditions, par leurs agents.

Nous rappelons, pour résumer, ce que nous avons développé dans ces leçons. A la combustion fonctionnelle correspondent toutes les manifestations saisissables de l'activité vivante. La synthèse organique, au contraire, a pour caractère distinctif d'être invisible à l'extérieur.

Au point de vue de leur nature chimique, les deux phases du travail physiologique sont exactement l'inverse l'une de l'autre : c'est l'analyse et la synthèse. Quant à leurs conditions, elles ne sont pas moins séparées : la combustion fonctionnelle peut s'accomplir *post mortem* et en dehors de l'organisme vivant ; les phénomènes de synthèse, au contraire, ne peuvent se manifester que dans le corps vivant et chacun dans un lieu spécial ; aucun artifice n'a pu, jusqu'à présent, suppléer à cette condition essentielle de l'activité des germes d'être à leur place dans l'édifice vivant.

La *rénovation moléculaire de l'organisme* est la contrepartie nécessaire de la destruction fonctionnelle des organes. Chez l'animal parvenu à son développement, chez l'animal adulte, les pertes se réparent à mesure qu'elles se produisent, et l'équilibre se rétablissant dès qu'il tend à être rompu, le corps se maintient dans sa composition et dans sa forme. Ces deux opérations de destruction et de rénovation, inverses l'une de l'autre, distinctes dans leur nature, sont absolument connexes et inséparables : elles sont la condition l'une de l'autre. Les phénomènes de destruction fonctionnelle sont eux-mêmes les instigateurs et les précurseurs de la rénovation matérielle qui se dérobe à nos yeux dans l'intimité des tissus, en même temps que les combustions, les fermentations se traduisent avec éclat par les manifestations vitales extérieures : le processus formatif s'opère dans le silence de la vie végétative ; le processus de destruction, au contraire, apparaît dans les manifestations de la vie fonctionnelle. La matière organique s'oxyde, s'hydrate, se sépare des tissus vivants, les abandonne ; mais, simultanément, ceux-ci attirent à eux, fixent et s'incorporent la matière inorganique du milieu ambiant. L'usure et la renaissance des parties constituantes du corps font que l'existence n'est en réalité autre chose qu'une perpétuelle alternative de *vie* et de *mort*, de composition et de décomposition, d'organisation et de désorganisation. Les dernières parties de

l'organisme, les éléments anatomiques, sont le siège de ce double mouvement d'*assimilation* et de *désassimilation*, d'*organisation* et de *désorganisation*, qui, considéré dans son ensemble, prend le nom de *nutrition*. Il serait peut-être préférable de réserver le nom de *nutrition* au phénomène de synthèse organisatrice, et de donner le nom de *fonction* au phénomène de désassimilation.

Nous ne rappelons ces faits de connaissance banale que pour avoir l'occasion de développer à leur sujet quelques vues que nous croyons nouvelles et qui sont relatives aux agents chimiques qui les mettent en œuvre.

D'une manière générale, nous distinguerons donc dans le corps vivant deux grands groupes de phénomènes inverses : les phénomènes *fonctionnels* ou de dépense vitale, et les phénomènes *plastiques*, d'organisation ou d'accumulation nutritive. La vie se manifeste par ces deux ordres d'actes entièrement opposés dans leur nature : la désassimilation, qui consiste dans une oxydation ou une hydratation d'une nature particulière et qui use la matière vivante dans les organes en *fonction* ; la synthèse assimilatrice ou organisatrice, qui forme des réserves ou régénère les tissus dans les organes considérés en *repos*.

Le système nerveux préside à ces phénomènes fonctionnels. Chez les êtres élevés en organisation, la manifestation vitale et par conséquent la combustion destructive qui en est la condition sont régies par l'appareil nerveux. On peut montrer que les fonctions des appareils nerveux sont réductibles à ces deux grandes divisions : le système de la destruction fonctionnelle ou de la dépense vitale, et le système de la synthèse organisatrice ou de l'accumulation vitale.

Thus, in the association between analytic phenomena and the manifestation of function, and that between synthetic phenomena and the accomplishment of nutrition, Bernard drew the antithesis between external manifestation of energy, *i.e.*, function, in association with analytic changes, and the internal replenishment of living matter in association with synthetic changes, *i.e.*, nutrition ; he did not lay stress upon the parallélism between action and analysis, rest and synthesis, which plays so important a part in the doctrines of the Prague and Cambridge Schools. By implication, although not expressly, he presents *function* as the effect of analysis in accordance with the axiom of energy transformation. And, so far from presenting nutritive synthesis as more

active in functional quiescence than in functional activity, he expressly says that nutrition is more active in the more active muscle. He assumes that both processes are under the control of corresponding classes of nerves promoting organic expenditure, or organic accumulation.

As regards the facts that may be appealed to in support of the modern theories of inhibition, they are as follows:—

1. Cardiac inhibition. (Weber.¹)
2. Vaso-dilatation. (Bernard.²)
3. The phenomena of colour-contrast, as presented by Hering.³
4. Dilatation of the pupil. (Grünhagen's experiments.⁴)
5. The excitatory and electrical effects and after-effects of anodic, and of kathodic influences. (Hering.⁵)
6. Biedermann's experiments on the claw of the crayfish, and on veratrinised frog's muscle.⁶
7. The positive electrical effect of vagus excitation on the tortoise's auricle. (Gaskell.⁷)
8. The failure of response in frog's muscle when its nerve is excited under certain conditions of strength and frequency of stimulation. (Wedensky.⁸)

All these subjects, with perhaps the exception of the last, are well-known physiological data, and need not be described here.

They have been thus simply enumerated as a memo-

¹ E. H. Weber. Wagner's *Handwörterbuch*.

² C. Bernard. "Influence des nerfs sur la couleur du sang," *J. de la Physiologie*, 1858.

³ E. Hering. "Zur Lehre vom Lichtsinne," *Wien*, 1874.

⁴ Grünhagen. *Pflüger's Archiv.*, vol. x., 1875.

⁵ E. Hering. "Zur Theorie der Vorgänge, in der lebendigen Substanz," *Pflüger's Archiv.*, "lotos," vol. ix., 1888.

⁶ Biedermann. "Weber das Herz von *Helix Pomatia*," *Wiener Sitzungsberichte*, 1883.

Biedermann. "Ueber Hemmungserscheinungen bei electrischer Reizung quergestreifter Muskeln," *Ibid*, 1885.

Biedermann. "Ueber die Innervation der Krebssehere," *Ibid*, 1887.

Biedermann (with Simchowitz and Fürst). "Zur Physiologie der glatten Muskeln," *Pflüger's Archiv.*, vols. xlv., p. 369, 1889; xlv., pp. 367, 398, 1890.

Biedermann. "Zur Lehre von der elektrischen Erregung quergestreifter Muskeln," *Ibid*, vol. xlvii., p. 243, 1890.

⁷ Gaskell. *J. of Physiology*, vols. vii., p. 451, 1886; viii., p. 404, 1887. Ludwig's Festschrift, 1887; See also Burdon-Sanderson, *J. of Phys.*, vol. viii., p. xxvii., 1887; Stewart, *Ibid*, vol. xiii., p. 154, 1892.

⁸ Wedensky. *Archives de Physiologie*, 1891, 1892.

random of the chief topics and data to be taken into estimation in forming any opinion with regard to the nervous mechanism of "phenomena of arrest," and in the generalisation of special "inhibitory" nerves. We may, I think, in the present state of knowledge, hold that the sum of these data goes far to establish the view that anti-motor are hardly less general than motor phenomena; on the other hand, considering the incomplete character of evidence that has been diligently sought for, we should be restrained from prematurely admitting the universal existence of specific inhibitory nerve-fibres analogous with the well-known cardio-inhibitory nerve-fibres.

With regard to theories as to the mode of action of admittedly "inhibitory" fibres, we are still in presence of two distinct tenable theories:—

1. *The old theory*, that an inhibitory nerve (vagus; chorda) produces its effect by acting upon "peripheral centres" (*i.e.*, terminal nerve-cells).

2. *The new theory*, that inhibitory nerves produce negative activity, by stimulating the positive or constructive phase of nutrition.

Comparing these two views, we cannot but recognise that the balance of probability is not heavily weighted in either scale. There is no cogent experiment to make us accept the new view. As regards the old view, we still have in its favour (1) that inhibition of action is an universal phenomenon of nerve centres; (2) that the old distinction between the "cerebral" or medullated nerve (vagus and chorda tympani) and the sympathetic or non-medullated nerve (vaso-constrictors and accelerantes cordis), associated with the well-known contrast between the actions of the two classes of nerves, is consolidated by the recent researches of Gaskell, according to whom the inhibitory or anti-motor fibre is medullated down to its distribution in distal ganglion-cells, while the augmentor or motor fibre is non-medullated in the nerve-trunk.

On review of the various positive items within our knowledge it appears to me (1) that as regards vascular and intestinal muscle the peripheral centre theory remains closer

to the facts than the trophic theory; (2) that as regards skeletal muscle there is no proof of the existence of inhibitory nerve-fibres.

The second of these conclusions is that which forms the subject of this paper. To consider it alone has been impossible; the question of the existence of anti-motor muscular nerves is closely linked with the doctrinal interpretations of which peripheral inhibitory phenomena are susceptible. Other problems of a similar and even less hopeful order irresistibly suggest themselves. To my mind one of the most pressing, and at the same time one of the least promising, as regards an answer justified by data, is the elementary question of the nature of processes and paths from cerebral to spinal centres. A spinal centre may be excited by way of a pyramidal fibre from the cortex; it can also be inhibited from the cortex. Can an inhibitory impulse and an excitatory impulse pass down the pyramidal tract in one and the same fibre, or do different fibres convey the two kinds of impulses? Here the balance is quite level, there is no "proof" in either scale, and about an equal weight of probability in both.

In conclusion, and to return to the particular class of phenomena studied in this paper.

(1) From the standpoint of Hering's induction, according to which an anodic effect or kathodic after-effect, is attended by diminished action and assimilation (= anabolism of Gaskell), while an anodic after-effect is attended by increased action and dissimulation (= katabolism of Gaskell); there can be no doubt that "anodic inhibition," as well as "kathodic excitation," are normal phenomena of human, as of all animal muscle and nerve.

(2) In any other sense, or, if Hering's generalisation be rejected, active arrest of action, as distinguished from passive cessation of action, *i.e.*, true physiological inhibition of voluntary muscle, has not yet been demonstrated. We have no right to believe that inhibitory impulses can pass to voluntary muscle, either by the ordinary motor channels or by any special inhibitory fibres; these last are not proved to exist.

These conclusions appear to me all that we may legiti-

mately infer from observations on the human subject. Even taking into account such data as have been obtained by electrical excitation of nerve and muscle of the lower animals, I do not think that we may at all safely admit direct inhibitory or anti-motor action of nerve upon muscle, as an article of general physiology; especially as regards the voluntary motion and cessation of motion in the higher animals, although it may be, and should be, recognised that positive evidence does exist on the lower animals which appears to favour such a doctrine.

But it is, I think, premature and hazardous to extend the notion to include voluntary motion of skeletal muscle. It is at most a working hypothesis to be verified or the reverse; and as far as experiments have yet gone, it has not been verified.

THE NERVOUS SYSTEM IN CHILDHOOD.

BY CHARLES MERCIER, M.B.

THE animal organism may be regarded as a mechanism propelled or actuated by energy contained within itself. The energy which it contains is disposed in two separate stores, available in very different ways. The one store is resident mainly in the grey matter of the nervous system, and is available from moment to moment to subserve the daily locomotion and other dynamic functions of the organism. In the execution of these functions this energy is expended, and, from time to time, the functions must be arrested while the store of energy is being renewed. The other store of energy is of far greater volume, and less circumscribed in its locality. It is diffused throughout, and resides in every organ and tissue of the body, having, however, its headquarters and main depôt in the nervous system.

This great and important store of energy is implanted in the organism at the time of conception, and is expended in carrying on development and life. It is under the operation of the forces drawn from this store of energy that the unfolding of the organism from its minute size and almost homogeneous structure at conception, to its adult stature and to the amazing complexity of full development takes place. It is by the gradual expenditure of energy from the same store that life is carried on after development is complete, and it is by the gradual failure and exhaustion of this store of energy that life at last comes to an end.

The energy that is stored in the animal organism may be likened to the coal that is stored in a steam ship. One portion of the coal is actually in the furnaces, and is

generating the steam by whose agency the functions of the ship—its locomotion, its electric lighting, cooking, &c.—are carried on. The other and greater store is in the bunkers, and its magnitude represents the distance that the ship can travel. The two stores bear from moment to moment no immediate and necessary relation to one another. The bunkers may be full and the ship may be capable of steaming thousands of miles, and yet the fires may be banked up and the engines at rest; and, similarly, the animal organism may be full of vigour, and contain the capacity of living a long and laborious life, and yet, at any given time, it may be at rest and conserving its energies for subsequent efforts. But although there may be no immediate and direct relation between the total store of energy and the amount that is at any one time being expended, yet, upon the whole, that ship which has the greater store of coal will not only steam the farthest, but will be able to maintain a forced draught for longer times, to proceed at a higher speed, and to exhibit a greater activity; while that which starts with an inferior stowage of coal will not only travel less far, but on the journey will be compelled to economise its expenditure, and will proceed more slowly. So with the living organism, the greater the initial store of energy, the longer the life; and not only will the life be longer, but it will be fuller of activity, more vigorous, more energetic.

If we apply the parallel to the cases of youth and age, we shall observe that at starting, when her bunkers are full of fuel to overflowing, the ship's furnaces are fed with the greatest ease, the supply being so ready to hand, and hence a full pressure of steam is at that time easily kept up; but that the farther she proceeds upon her journey and the more coal is used, the emptier do the bunkers become, and the farther has the fuel to be fetched; hence the furnaces are fed more slowly, the full pressure of steam is less easily and less often attained, and the visible signs of activity diminish. Similarly, in childhood the bodily powers are always readily available, activity is great, and is easily evoked; while in the later life activity is less eager, less spontaneous, and seldom attains the same high level. At the risk of becoming fanciful one is

tempted to carry the parallel yet further, and to note that just as in the one case, as time goes on, the boilers become encrusted and the machinery worn, so that, even with the same expenditure of coal, a more delayed and less efficient activity is attained; so in the other case, as the store of energy becomes depleted, the tissues degenerate, and even with the same expenditure of energy, activity is less promptly evoked, and when evoked is less efficient. As the returning ship with her boilers encrusted, her pipes leaking and her bottom foul, cannot compete with the ship just commissioned, whose machinery has just left the maker and whose hull the dock, so the man of mature age is incapable of the activity of the boy not yet out of his teens.

By as much as the child is nearer to the starting-point of its existence than the adult, by so much will the child's reservoir of energy be more replete and better stored than that of the adult. The child has proceeded a shorter distance on the journey of life and development; it has consumed less of the fuel with which it started; it is capable of proceeding farther, and it is at the time being proceeding with a stronger and more vigorous impetus. This is the first difference between the child and the adult.

The next difference is that, not only does the child possess a more copious store of energy, but this energy is more readily and immediately available, it is nearer the surface, as it were, and more easily evoked.

As the child has proceeded less far along the path of development than the adult, it follows that its nervous system has not yet attained the characters which are acquired in the later stages of development. These characters are definiteness, fixity, and complexity of organisation.

Not only is the child's nervous system less completely developed than that of the adult, but further, it differs most conspicuously and most importantly in being in a condition of rapid change. The one is in process of rapid and momentous development, the other has attained nearer to completion, and its changes are less extensive, less important, and more gradual.

Finally, the nervous systems of the child and the adult differ, not only in structure, in progress and other intrinsic qualities, but they differ very importantly in the forces which act upon them. The adult is comparatively exempt from stresses arising from tumults in his own organisation, to which the child is specially subject, and is obnoxious to many stresses arising out of surrounding circumstances, which are harmless to the child. We will now take these several differences seriatim and examine them in detail.

The first difference noted was the more abounding energy with which the child is animated. It is nearer to the starting point of its existence, its development and growth are incomplete, it has travelled but a short distance on the journey of life, and it contains within itself the vast store of potential energy which is capable of carrying it through these intricate and exhausting processes. Each step of development, each increment of growth, each year of life, is gained, is passed through, by the expenditure of a portion of this store of energy, and hence it is evident that the further that development, growth and life have proceeded, the more of the original stock of energy has been expended, the less remains in store at the service of the organism for the supply of its future needs.

On the other hand, the less far the organism has proceeded along the path of life, the more copious, abounding, and overflowing is the amount of energy at its service, the greater the pressure of the forces that are urging it along this path, and the more powerful the impetus with which it is proceeding.

Now, the path of a moving body is direct in proportion to the vigour of the impetus by which it is impelled, and this law is as true of a living organism moving along the path of development under the influence of its genetic impetus, as of a projectile moving through a retarding medium on the propulsion of a charge of gunpowder. In the latter case we know that the more forcible the impetus that the projectile receives, the greater is its penetrating power, that is to say, the less easily is it turned out of its

course by the obstructions that it may meet with. And precisely the same law holds in the former case also. In these considerations we find the explanation of the fact that the young are less susceptible to many noxious influences than the old. A lad can prolong his bathe in the sea for a length of time which would bring his father into a state of collapse. He can commit with impunity a thousand indiscretions in diet, in exertion, in exposure, in excess, which would have serious consequences if indulged in by a man of mature age. So a convulsion in a child has a far less important significance than the same symptom in an adult. So, too, the recuperative power of youth in nearly all maladies is much greater than that of mature life or of age. It is one of the commonplaces of prognosis to speak of the advantage to the patient of his youth, the disadvantage of age, as witness the relative gravity of a fractured thigh or an attack of acute pneumonia at these several periods of life. All these advantages of youth arise from the greater store of energy that is possessed in early life. We may compare them to the superior stability of a spinning top during the early stages of its career, when it will sturdily right itself after a momentous disturbance; and the precariousness of its balance in its later or wobbling stage, when the energy that actuates it is nearly dissipated, and when a trifling touch is enough to upset it altogether.

To its nearness to the point of its origin, and to the small influence which the friction of circumstances has as yet exerted upon it, the child owes its freedom from the whole group of disorders of degeneration—from the scleroses, the atrophies, the apoplexies, the deteriorations of connective tissue, of blood-vessel, and of nerve tissue also, which tend to appear as life advances, and as the initial store of energy becomes dissipated.

While the superior impetus of its progress through life, and the greater and more readily available store of energy that it contains, thus confer advantages on the youthful organism, they at the same time bring with them certain inevitable disadvantages. That energy which is so ready to hand and so easily evoked, at the normal requirements of

the organism, is also more ready to break out on the summons of pathological irritation; and the discharge, which is always plenary, may easily become excessive. Hence we find that in childhood the irritation of a budding tooth or of an undigested meal is enough to evoke a convulsion. Thus, too, we explain the much greater frequency of chorea in childhood than in adult life; for this reason we find certain convulsive disorders, such as laryngismus stridulus and tetany, confined to the early years of life.

The next point of difference between the immature and the adult nervous system lies in the incompleteness of the former. The infant is born with a nervous system in which all the endowments of the adult are potential, none but the very lowest are actual. The several functions are not yet distributed among definite structures. The structures, no more than the functions, have yet come into existence. The great central nervous mass is, as to the internal arrangement of its higher regions, without form or definite arrangement. It is a quasi-homogenous aggregation of explosive material, in which definite lines of activity have not yet made their appearance. Until the higher regions of the nervous system are organised, their functions cannot of course exist, and hence we find that in the infant so primitive a function as the control of the sphincters has not yet been acquired. When the function is acquired, however, it takes time to perfect—to consolidate—to attain completeness of organisation. The first efforts at speech are tentative, and a word gained yesterday may to-day be impossible of utterance. So the control of the sphincters may be lost for a while—may undergo several temporary lapses before its embodiment in the nervous structure becomes complete; or it may be efficient during the day, when all the higher nerve regions are acting, but become evanescent at night, when these centres are in repose.

From the inferior definiteness of the child's organisation results the inferior definiteness of the manifestations of its disorder. The highest nervous regions have not in the child yet come into existence as such; but the material out of which they are to be formed—the amorphous intercellular

and interfibrillar ground substance, which is the matrix from which they will in course of time be differentiated—already exists in considerable quantity in that part of the brain in which the highest nerve regions will eventually appear. It exists, and it is to some extent capable of acting, but since its structure is unformed, since its elements are undifferentiated, its paths of discharge unexcavated, what action it may have on the inferior regions of the nervous system and on the body at large, can be only vaguely characterised—cannot be definite—must be wanting in formal characterisation. For this reason all nervous maladies dependent on disorder of this region are wanting in the precision of character that attend them in adult age. We never see in an infant the definite opening and orderly march of the convulsions of Jacksonian epilepsy. What convulsions they suffer are universal and uniform. They are not sufficiently differentiated either in time or space to admit of description, either as to their progress from beginning to end, or of the parts of the body involved. They consist merely of a general, brief, uniform overflow of nervous energy, resulting in an universal, brief, and uniform muscular spasm.

These remarks apply, of course, to the superior nerve regions only. Since the inferior regions are already in childhood well differentiated, damage to these regions, if sharply delimited, may produce definitely localised defect, and hence becomes possible the form of paralysis known as infantile.

In these considerations we get an inkling of the reason why insanity—acquired insanity—is never seen among children. Insanity depends on disorder of the highest nerve regions, and in the child the highest nerve regions have not yet come into existence, and therefore cannot be disordered. That the child should suffer from disorder of the inferior nerve regions which are for it temporarily the highest—the highest, that is to say, that have yet become developed in it—is unlikely for this reason; that disorder does not occur under ordinary stresses unless the structure of the nerve regions is originally defective. Now in childhood extraordinary stresses are so extremely rare that they

may be neglected ; and we have only to consider the reason of the rare occurrence of insanity under ordinary stresses, that is to say, we have to ask why the (temporarily) highest nerve regions are so seldom wanting in stability. The answer is to be found in the considerations already advanced. The impetus in the course of development that is given at conception is strongest at the outset and gradually weakens with the friction of the organism against its circumstances. Hence the earliest stages of development are the most certain to follow in the normal course, are the most difficult to disturb from that course, and produce structures of the firmest stability. The inferior layers of the highest nerve regions, which are in the child temporarily the highest, are developed at an earlier period of life, at a time when the developmental impulse is still powerful, and its capacity of driving the organism directly forward in the normal course of development is great and unexhausted. Structures formed at this period of development attain a high degree of stability and are not liable to disorder under stresses of ordinary severity, and thus acquired insanity is likely to be in childhood extremely rare.

Again, if insanity is, as I have elsewhere stated it to be, a disorder of the process of adjusting self to surroundings, there is another reason why it should be rare in childhood : for at that time of life the individual is placed in circumstances of extreme and artificial simplicity. The surroundings are supplied, as it were, already adjusted. But little adjustment on the part of the individual is needed, and if much is absent its absence is unnoticed.

While the nervous system of the child is less definite than that of the adult, it is also, by reason of its immaturity, less fixed in its organisation, less rigid, more plastic. There is less of finality in its arrangements, they are more easily modifiable. On this greater plasticity of its nervous organisation depends the greater capacity of the child to be modified by the influence of its surroundings. It is more trainable, more teachable, more readily assumes new characters, acquires new capabilities. Necessary as this plasticity of structure is to the development of the child, it

brings with it its inevitable disadvantage. If useful acquirements are at this period of life more readily learnt, and beneficial habits more readily attained, on the other hand, other acquirements and other habits of less beneficial character are also assumed without difficulty. A structure which is more modifiable and more plastic, is more susceptible, not only of beneficial influences, but of harmful influences also; and hence we find, not only that children are liable to nervous disorders from which adults are free, but that all stresses to which both are subject produce a severer proportional effect on children than on adults. The shock and sudden derangement of nerve molecules that attends a fright, will in an adult cause only a start and a cold sweat, while in the child it may so obstruct customary paths of discharge as to produce a stammer, or may give rise to so widespread and excessive a running-over of discharge as to produce chorea. A blepharospasm, which in an adult occurs as a transient affection, may easily in the child become a permanent habit.

The third indication of the incompleteness of the child's nervous organisation is its inferior complexity. Many of the capabilities of the adult have not yet been acquired by the child, and consequently the nervous arrangements which underlie these capabilities—whose activity constitutes the capabilities in question—have not yet been organised. Its nervous organisation is comparatively far less complex than that of the adult. Now *cæteris paribus*, the more complex a mechanism, the more easily is that mechanism deranged in its working; and the simpler it is, the more difficult is it to put out of gear. This is one of the reasons why there are whole classes of nervous maladies to which the adult is subject and from which the child is free—the other reasons are considered in their places. Among the maladies in question are those of hysteria, of trance, epilepsy, and the group of “functional impotences,” writers’ and other cramps.

The next difference between the nervous systems of the child and of the adult is that the former is undergoing rapid development—is in a state of constant transition—of

active change, while in that of the other the changes are less important in character and slower in their course. In the child there is going on a rapid acquisition of faculty, and in the older child a rapid development of feeling, both implying large and rapid developments of nerve tissue. Now, every structure in which rapid and extensive internal changes are proceeding is more liable to be disordered by incident forces than one whose component parts are in relative rest. A State which is undergoing internal revolution is more at the mercy of an invader than one which has a fixed and stable government. When iron is heated to redness—when, that is to say, its integral parts are in a state of more rapid and wider movement, it more readily changes its shape under the hammer. When a regiment is deploying—is changing its formation—then is the moment to direct a charge against it, for then the attack is more likely to be successful. And when the nervous system is undergoing rapid and extensive changes, then the incidence of forces that would be innocuous at other times is likely to produce disorder. Here, then, is another reason for the liability of the child to suffer disorder from causes which do not produce disorder in the adult. Hence certain nervous disorders are peculiar to childhood or are much more frequent at that time.

Lastly, the nervous system of the child differs widely from that of the adult with reference to the disturbing forces to which it is subject. Each is subject to influences from which the other is free ; and, generally, the immature individual is more subject to disturbing influences arising from within its own organisation, while the adult is more subject to influences impinging from without.

The body of the child is growing in bulk, and is attaining at the same time a higher standard of complexity of organisation ; and both these processes are taking place rapidly and somewhat tumultuously. The entire organisation of the child is in a condition of rapid and momentous change. Now every change that takes place in the body at large produces an impress on the nervous system. Every bodily process initiates currents in the nerves of the part in

which the process occurs : currents which flow to the central nervous masses and there produce effects. The more numerous, the more momentous, the more rapid the changes in the body, the graver and more powerful are the influences to which the central nervous system is subject from this source ; and when the bodily changes are so rapid and so momentous as at some of the periods of growth and development, the impress upon the central nervous system thus originating may be so grave as to be an efficient cause of disorder. In this we find another and the chief reason of the prevalence of hysteria in youth. In the adult, as the changes proceeding in the body at large are comparatively few and slow, so the action on the central nervous system that arises from this source is comparatively inconspicuous and productive of no marked result. Hence, disorders arising from these internally initiated stresses are rare in adult life, common in youth.

While the stresses generated within the organism itself are more severe in the child than in the adult, on the other hand the child is comparatively exempt from stresses of external origin, to which the adult is specially obnoxious. From the burden of earning his living and the anxieties attendant thereon ; from the tribulation of producing and rearing offspring, and the tempestuous experiences that precede and accompany these processes ; from the exultations and disappointments that attend the strivings of ambition, the child is exempt, and thus is free from so many sources of, and provocations to, disorder.

Thus we find, upon a review of the whole circumstances, that in general terms, the nervous system of the child is more easily disordered in its action than is that of the adult ; but that, having suffered disorder, it has a stronger tendency to return to the normal condition. We find that the manifestations of nervous disorder in youth are apt to be less definite in form than in adult age ; and that the deranging agents at the two periods of life are widely different.

THE TRAUMATIC FACTOR IN MENTAL DISEASE.

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THIS article begins with some general considerations bearing on the subject ; and in the second place deals with its special clinical and pathological aspects.

1.—GENERAL CONSIDERATIONS.

(a.) I include all injuries, produced by external violence, affecting the nervous system so as to become a factor in the production of mental disease. Of these, the most important are *direct* injuries to the head, but to limit our subject to these, as some would, is narrow and unscientific, inasmuch as precisely the same mental result and the same lesion may follow cerebral disturbance or injury produced *indirectly*.

(b.) Nor do I (as many do) include insolation here, as a traumatic factor. Clinically similar as are some insolation cases to the traumatic ones, the two appear to be properly kept separate.

(c.) The surgical aspects of the subject are not dealt with here—*e.g.*, questions of surgical operation, traumatic fever, or the symptoms of brain-compression or concussion.

(d.) The traumatic factor may be predisposing or exciting ; may be co-operative in preparing the soil favourable to insanity ; may give rise to mental and other symptoms at the time, but these passing off, the incident may leave some impress conducing to mental weakness or instability, or may leave behind it some organic change which, increasing or undergoing a natural succession of phases, may eventually lead to mental failure or disturbance. Or the mental disease may spring directly and without interval from the occurrence of the injury ; or after a shorter or

longer interval devoid of symptom, or marked only by slight indications of threatening nervous and mental failure or perversion.

Thus the traumatic factor may be simply predisponent. Or, at the opposite pole of action, it may be the direct excitant of an insanity already about to appear, the particular spark that happens to fire the prepared train; or it may act on a less fully prepared condition, and modify the course and symptoms of the insanity it precipitates; or it may lead to the formation of a neurasthenic or hysterical condition, and coincidently or thereupon may come psychic disorder; or to the setting-up and development of a special traumatic neurosis, and of a morbid psychosis; or there may perhaps be, primarily, coarse brain-damage; and either secondarily, or without such damage, organic and often progressive destructive brain-disease. These last two groups include the cases of insanity more completely and characteristically of traumatic nature.

(e.) The traumatic factor frequently is but one of several, not often do the causes of insanity act quite singly, several factors usually co-operate; the traumatic factor often modifies, and is modified by the operation of other factors.

(f.) The injury may produce a functional disturbance or suspension only; or, on the other hand, the mental disease may be symptomatic of demonstrable, and often gross, traumatic organic lesion of the brain.

(g.) The *immediate* morbid conditions arising out of injury, and constituting a factor in the production of mental disease, may be molecular perturbation of brain (concussion); or contusion, crush, rupture of its substance, or hæmorrhage into its tissue or into the meningeal spaces; or vaso-motor effects of damage to brain, cord, or sympathetic nervous system. Some of these conditions may promptly produce compression and local anæmia or œdema of brain.

And in these cases the *secondary or remote* morbid conditions of brain, &c., due to injury and favouring abnormal psychosis, may be slow nutritive alteration, inflammation and its exudations, suppuration, hydrocephalic states, local bone disease, chronic inflammation and sclerosis, secondary

degeneration, the absorbent changes following destructive hæmorrhage softening inflammation or ischæmia of parts of the brain-substance; such ischæmia being producible by arterial thrombosis, or by compression of blood-vessels as by clot or exudation. Or tumour-growth, originated by injury, may in its turn foster mental disorder; or press for mental failure through cerebral compression and disorganisation.

(*h.*) Obviously, the symptoms and the whole course and tendency of the resulting psychosis, other things equal, must vary with the infinitely numerous varieties of kind, extent, and degree of severity of the injury produced; and with the nervous and mental tendencies of the individual. So must they vary, other things equal, with the part or parts of the encephalon functionally affected in any way, disordered in circulation or nutrition, or the seat of damage. Mere mention of this must suffice;—the whole wide subject of the localisation of the functions of the nervous system is involved here.

Especially in the functional cases have I noticed the frequency with which the back of the head has been the part struck.

(*i.*) Speaking generally, the *age* of the patient is of some effect; the tendency at each age of life being to the production, by injury, of the particular forms of insanity to which each such age is most liable.

(*j.*) The tendency to insanity of traumatic origin is greater in civilised than in savage man—is enhanced in the subjects of the neurotic and insane diatheses; or in those of sensitive, irritable, excitable, impulsive, wayward nature and disposition; also in those prone to sexual, alcoholic or narcotic excesses; or enfeebled by disease, mental strain, anxiety, insomnia, or privation. Particularly have I observed precedent alcoholic intemperance or syphilis.

(*k.*) When insanity comes on some considerable time, or long, after the injury, certain conditions are apt to mark the interval between the cessation of the immediate effects of the blow and the advent of the morbid psychosis. Such are the easy production and exaggeration of the effects of alcohol,

narcotics, coition, extreme heat, mental over-work or agitation, the troubles of life and knocks of fortune. Even the excitement of physical exertion occasions an undue cerebral effect and mental commotion.

A change of character is frequent ; and usually is in the directions of unwonted impatience, irascibility, furious outbursts, a quarrelsome, overbearing brutality ; or moody, unsocial taciturnity and suspicion ; and with these, or alone, general mental failure, or gaps in memory ; cerebral fatigue on the least mental exertion or strain of attention ; or mental confusion—a dazed, bewildered condition ; or emotional dejection and hypochondriacal notions ; or an uneasy nervous state, a general *status nervosus* ; bad dreams ; ready addiction to alcoholic indulgence or sexual pleasures.

These conditions may deepen into the prodromal stage of the coming psychosis ; or, when this is reached, we may find suicidal and homicidal impulses, or an expansive phase, and usually insomnia ; or preliminary to the onset of the overt psychosis there may be disquietude, tremor, headache.

Various sensory and sensorial disorders and defects are frequently precursory :—pain in the head, especially at, or chiefly at the seat of old injury, or tenderness there ; or general cranial pain ; or numbness and various paræsthesiæ. The disorders of special sense observed in different cases are too many to mention here ; and they, like the symptoms above-mentioned, vary from the products of focal lesion to the abounding severe symptoms of traumatically roused neurasthenia and even hysteria.

As for the motor conditions which may arise, early or late, there may be found all forms of paralysis, paresis, tremor, spasm, *convulsion*, contracture, chorea, ataxia. Vertigo may occur. These, however, more often belong to the later time of actual mental disease.

At first the symptoms may be slight, but severe later on ; or symptoms severe at the onset may give place to milder and recovering ones.

(l.) Insanity traumatically occasioned may come on immediately or rapidly after the injury, or months, or many years later. It may be related in all sorts of ways to other

forms of insanity; nor is there space here to discuss these relationships with any fulness.

(*m.*) The prognosis is moderately favourable in some of the functional cases; unfavourable in the organic. Intensely severe symptoms at the onset often permit a more favourable prognosis than those beginning slowly, and becoming worse gradually. Epilepsy, or convulsion, is an unfavourable omen.

(*n.*) In some, clinically, so-called functional cases, as well as in the organic, there may really be some tissue-injury, produced by the violent vibration and propulsion of the cerebro-spinal fluid (in concussion), demonstrated by Duret. Or, again, there possibly may be breaking-up of the nerve-fibre myeline, and formation of bodies related to colloid ones (Miles. See BRAIN, 1890, p. 224). Or, when of the ordinary type, limb-paralysis, occurring in some so-called functional cases, on the same side as the cranial injury, may really be due to some damage of the opposite hemisphere of the brain, by counterstroke.

(*o.*) In the later-arising and more typical cases the psychosis is built upon the foundation of a traumatic neurosis—a neurotic state partly described above—springing out of the effects of the original injury.

(*p.*) *Pathologically*, from the shock of a blow may come singly, or in succession—(*a*) jar, shake (concussion as a cause), with its hypothetical molecular changes of structure, as a *primary* effect, and with it more or less suspension of some functions of the brain and spinal cord; (*b*) the basis of traumatic cerebral and spinal neurasthenia, or other neuro-psychosis—namely, some molecular alteration of those organs; and (*c*), as *secondary* or *late* effects; subacute or chronic demonstrable organic brain-disease.

Severe crushes of brain take a different line of morbid process.

Moreover, (1), the original injuries may be to the skull-cap, and the brain be secondarily affected by these. Or (2), the brunt of the stroke may fall on the intracranial blood-vessels, producing rupture, &c. Or (3), the brain-substance itself may suffer primarily and chiefly.

In the third and last case, there may merely be functional disorder and molecular change of the brain ; or, later on, it may become compressed by extravasation or by inflammatory exudation ; or may undergo profound nutritive alterations ; or, on the other hand, may incur gross material primary damage.

Clinically, in the *adult*, we see four great branches diverging from the traumatic stem :—One consisting of functional neurotic states with mental symptoms, chiefly traumatic neurasthenia and traumatic hysteria. Another consisting of ordinary and definite forms of psycho-neuroses—functional mental perversions of the more simple type. Another constituted of those usually of hereditary mental degenerative type, but now either of direct traumatic origin, or merely favoured in formation by injury. And another consisting of mental and other symptoms, severe and very often incurable, tending to dementia and death, and based in damage, or in severe organic brain-changes starting from injury.

II.—SPECIAL CLINICAL AND PATHOLOGICAL ASPECTS.

To begin with, the influence of external violence may be exerted even during *intrauterine life*. Jars, shocks, commotions, explosions, or other injuries, acting through the mother, and affecting the germ-layers of the embryo, or incident on the foetus, are sometimes the source of congenital mental defect, or future mental disorder, both based in cerebro-spinal non-development, malformation, or disease, of traumatic origin. Numerous experiments on the embryos of lower animals show the immense effect of injury in this direction.

Next, when born into the world, man, from the cradle to the grave is pursued by a liability to the effective incidence of the traumatic factor of mental disease. In the first place, the act of *birth* itself—in civilised races especially—is full of danger to the infant. Difficult labour—dystocia—without instrumental aid, easily produces various detrimental cerebral effects, including structural brain-damage ; instrumental aid

often is obtained at the cost of bone- and brain-injury, perhaps extrinsic to the occasion.

And at the next stages of life—in *infancy* and *childhood*—falls and hurts lead not seldom to concussion of brain; or to its contusion, hæmorrhage, crush, or compression—although if this last is due to depressed bone the comparative elasticity of the brain-pan and the circulatory activity and the plasticity in early life, may now permit of a natural and sufficient restoration of the cranial conformation, and restitution of the position and integrity of the brain, without the aid of surgical art.

Congenital injury, or that coming in early life, tends to produce idiocy or imbecility:—often with these are convulsions—usually classed as epileptic, but not seldom more properly epileptiform—talipes, hemiplegia, contracture, wasted limbs. Choreiform or else athetotic movements often attend such cases. Ill-temper, quarrelsomeness, readiness to assault and destroy, come in paroxysms, and epileptoid automatism may be one of the clinical phenomena. The tendency is to impulsive excitement and destructiveness, often of epileptic or epileptoid origin; progressive mental degradation and death. At the necropsy, we find traces of old meningeal hæmorrhages, local wastes, and destructive lesions of brain-surface; but more often a unilateral hemispherical cerebral atrophy, more obvious in some lobes than in others.

Some cases, starting from injury in childhood, take convulsion as the great phenomenon, become confirmed “epileptics”—at least, are so classed—and have all the clinical phenomena—mental failure, welling-up of morbid ideas, violence, irritability and automatism, of the epileptic; although sometimes the condition is only epileptiform. And after death, the only naked-eye morbid brain conditions may be some brain-wasting, and some developmental irregularities of gyri and sulci in the posterior half of the brain, as in *Case No. 1*.

In the next stage of life—that of *youth*—may come seizures in which, after a depressed prodromic stadium, the subject is sleepless, boisterous, loquacious, noisy, violent,

mischievous, destructive; and this either in recurring seizures or subcontinuously. Such cases may recover; or may become chronic, take convulsions, and die.

Other cases, with injury a factor in youth, and allied to the idiocy-imbecility group, are examples of so-called "moral insanity," and of various other forms appropriately termed of the hereditary mental degenerative series, such as insanity from impulsive ideas, original mental twist, hebephrenia and paranoia.

And next, in *adult* life, more especially, is a great variety of cases. Some of these occur also in youth. According to my observation, in the *adult*, and especially following *cranial* injury, there are chiefly four great groups of mental disorder:—

(1) One consisting of definite ordinary, although perhaps modified, forms of functional mental perversions of the more simple type (psycho-neuroses).

(2) A second constituted of paranoia and its immediate congeners, without or with obvious organic change; also—

(3) The third, comprising mental and other symptoms dependent on severe, traumatic, organic brain-disease and alteration, whether secondary, or including also the primary damage of the brain. Focal brain lesions, diffused brain-disease; organic dementia, epileptoid states, general paralysis and conditions akin thereto; and

(4) The fourth, consisting of certain functional neurotic states, with mental symptoms, chiefly of the type of traumatic neurasthenia and hysteria. These may incidentally crop up in any of the other groups.

The second and third groups comprise the cases more fully and characteristically of traumatic nature, as morbid psychoses.

I.—In the *First* great group, consisting of functional mental disorders of traumatic origin in adults, we take up ordinary, although perhaps modified, psycho-neuroses.

Cerebral and Mental Automatism. Case 2.—Cerebral automatism, stuporous form of hallucinatory insanity, emo-

tional depression. After a cranial hurt, by falling on ice, were automatism and profoundly obscured consciousness for a time during which the patient made his way (about 300 miles) to London, but afterwards retained no recollection of the journey or of how he came; there being a complete mental blank from the moment of the blow on the head to that of finding himself in bed in a London infirmary. The head continued to be painful, especially on the right side, and for a time the right shoulder and right side of chest. Out of the automatism, he passed into a condition of vacant immobility, alternating with restlessness, bursts of weeping, and once an explosive outbreak of transitory destructiveness; and thence into a condition, between hallucinatory and stuporous insanity, characterising the group linking and forming the transition between the typical forms of those two disorders; there being connected dream-romances, or vivid hallucinations of sight, hearing and smell; some stupor; occasional emotional depression and weeping; delusions of annoyance and torment in connection with the hallucinations, which also were of hostile import. An antecedent influence was the disappointment of failure in business.

Case 3.—At the other extreme, as regards rapid or slow evolution of the symptoms, is the next case, in which—long after very severe cranial injury, immediately following intense symptoms, including prolonged coma, treated at the London Hospital, and incurred thirteen years before admission to the asylum under my charge—came pain and tension of head, increased by warmth or by stooping, constrictive head-pain, bad dreams, sleeplessness, strangeness of manner and expression; at times apparently hallucinations or illusions of sight and hearing; kleptomania—the thefts, however, being performed quasi-automatically, and the patient repeatedly finding the stolen articles at his home without being able to tell how they came there. A somewhat self-willed patient, at times voluble, excitable, quarrelsome; occasionally roused to explosive anger, and, then, becoming abusive and insubordinate.

In these cases were hallucinations. But characteristic examples of traumatically induced

Acute Hallucinatory Insanity, with unsystemised delusions, are such as that of *Case 4*, with hallucinatory insanity from injury to the head occasioned by a blow from a poker over the back part of the vertex (at about the posterior fontanelle), the loss of blood being considerable, and there being the effect of previous drinking habit, and the mental agitation of a quarrel with, and separation from, the man with whom she had cohabited. Vivid hallucinations of sight and hearing, and of hostile import arose; also delusions of being jeered at, tried for crime, accused of prostitution and of suffering from venereal disease, delusions of impending evil and of the complicity of persons about her in an intended perpetration on her of (physically and mentally) cruel brutality. These, conjointly, led to dejection, a suicidal attempt, and violence to others.

Then, again, in *Case 5*, following in the wake of syphilis and sexual excess, a blow on the top of the head was promptly succeeded by hemiplegia, hallucinatory insanity, great lack of emotional control, delusions of wires being attached to the ears and mouth, of annoyance, of prevention from obtaining a livelihood, of persecution. The symptoms tending to the paranoia group, in character

Melancholic or Depressed Cases.

As *Case 6*, where cranial injury, arising out of a quarrel, was followed by a dazed, confused, self-worrying state; sadness, dejection, inability "to content himself" or to fully "know what he was doing"—a state of simple melancholia, with several suicidal attempts. Termination: recovery.

Or, as *Case 7*, where occipital injury in an alcoholised person is a factor. Melancholic notions of wickedness and worthlessness; vivid hallucinations of sight and hearing as to hell and heaven, as to persons being burnt, drowned or confined. Assertion that he himself had been drowned. Morbid fears of falling forward or backward (somewhat like hypsophobia). The symptoms not of purely alcoholic type. Termination: recovery.

Or, as *Case 8*, in which, at and around the bump, at the vertex, of a cranial injury incurred fifteen years previously,

come a recrudescence of pain and a feeling of numbness. Melancholic delusions of great wickedness; morbid fears; and hallucinations or illusions of sight, hearing, taste and smell. Termination: recovery.

In *Case 9*, aged 50, injury over the upper and anterior part of the left parietal bone, and encroaching on the frontal, had been incurred many years previously, and, latterly especially, he had been very easily affected by drink, to which he was moderately addicted. He was melancholic and demented, there being considerable failure of memory, emotional dejection, dread of vague impending evil, hallucinations of hearing and, for a short time, of sight.

II.—*Paranoia* is the chief form in the *Second* great group; and tending that way are some cases of unsystematised delusion with hallucination. In some cases the cranial injury appears to be a factor acting in early life and modifying the mental state so as to assist in forming the natural bent to insanity—i.e., creating a latent susceptibility to the insanity. Unsystemised delusional insanity with hallucinations at first, may or may not gradually be replaced by the systemisation of established paranoia. Thus (*Case 10*) unsystemised delusions as to hostility against the patient, as to stuff being put in his food, and his being accused of murder, and correspondent hallucinations of smell, taste and hearing—coming on at the age of 25, had been preceded by sequelæ of severe cranial injury at the age of 11.

In this group may also be symptoms of organic brain and cord disease of traumatic origin.

Case 11.—Age 47. Fell on an iron deck, over two years ago, and injured his occiput, was stunned for a few minutes, and found his fingers and toes “numb.” Worked for the greater part of a year afterwards; but, and very noticeably so after the expiry of six months, his sight became “misty.” He became shaky, had to give up work, was treated at Guy’s Hospital (where he had anæsthesia and analgesia of the feet, if his description is correct), and since then in a workhouse and in the asylum under my care. He is depressed, half weeps; has delusions that his friends

are all killed, that he is made incapable of knowing anything, that he is to be killed for committing murder and cutting up children, that false accusations are made against him, that—through the windows—persons tell him he will be burnt up. Hallucinations of taste and smell co-exist.

Forehead and eyebrows raised and corrugated, lower face somewhat flabby; the eyelids blink and twitch; pupils dilated, the right one the larger and sluggish to light. Speech pausing and hemming. Gait slow, with short unsafe steps, and somewhat staggering; stands shakily with *tremor a debilitate*, knee-jerk nearly absent: the patient, nearly blind, describes pains of somewhat fulgurant character. The moderate head-pain is chiefly frontal, when present.

Some become moody, solitary, moping, impatient, bearish, ill-natured, irritable. Then the excitement waxes, is accompanied by threatening and violent language, and may pass into an acute excitement with suicidal attempts and homicidal assaults, and perhaps convulsions. The acute symptoms, passing off, may leave a bitter, resentful, suspicious, morose, unfriendly, unsociable, taciturn, reserved condition and demeanour; and delusions of conspiracy against the patient.

Or, an early and longer or shorter period of more or less excitement may subside into chronic mental perversion; marked by the dangerous, often homicidal, suspicious, irascible, irritable state of the subject; the symptoms in my own experience being of the depressed, hypochondriacal, persecutory, aggressive type; and expansive symptoms coming almost only in the groups next to be described. But some cases, apparently to be classed here, and described by others, present a commingled aspect of the delusional phenomena—oppressed and expansive—those of suspicion mingling with the proud and egoistic ones. Fits of ungovernable fury may paroxysmally add tumult to the scene—fury either of epileptic basis, or not. The end is usually dementia and death.

Or, the change of character already described in the preliminary part of this article may be followed by, or by in-

creased, headache, insomnia, irascibility, failure of memory, or a temporary and somewhat partial darkening of it especially apt to come in connection with "fits"; suspiciousness, homicidal and suicidal impulses; or homicidal assaults under delusion as to the identity of the assailed, as in a case by Brower. These symptoms may remit sharply.

Persecutory or hypochondriacal delusions are frequent; those of poisoning of, or infidelity to, the patient, are not infrequent. In some at least, of these latter cases, the traumatic factor is complicated by the effects of drink. Thus, in *Case 12*, where insanity had followed a severe injury to the occiput seven months before the patient's admission, there had also been moderate addiction to alcohol. The patient had delusions of the infidelity of his wife with a youth next door, of the (real or imaginary) "tappings" heard by him being the signals between the guilty couple; and so intensely suspicious was he that every little circumstance was twisted into support of that view. Becoming miserable about it, he wanted his wife to drown herself together with him; bought a pistol to shoot both her, the youth, and himself; frequently threatened his wife's life, and once attempted to strangle her.

Tendency to alcoholic excess, to dissolute life in any form, moral perversity, brutality to members of family or household, and, later on, graver outrages, brutal and violent impulses, and, perhaps occasional outbreaks of acute mental excitement, mark many cases.

The course is long and changeful, fluctuating from lucidity, or a merely hypochondriacal state, to extreme paranoiactal perversion; the tendency is to fatuity and death. Insomnia, noises in the ears, dulness of hearing, vertigo, local pareses, visual and other hyper-, par-, and an-æsthesias, headache of special characters, are frequent. Symptoms arising may be neurasthenic or hysterical. Marcé described a badly defined form, with irregular alternations of stupor, agitation and imperfect lucidity, without systemised delusions.

Senile Dementia or *Senile Insanity* may be modified and precipitated by injury. Having a distinct basis in organic

cerebral disease it forms a sort of link between this group and the next group, and might be described with the latter. Yet its organic basis is independent.

Case 13.—Aged 71, was admitted after making several recent suicidal attempts. Many years ago a falling cask struck him on the top of the head ; seven or eight years ago he was beaten about the forehead ; and some months ago his head came sharply into collision with a fireplace. To these injuries he attributes the noise in the head of which he makes grievous complaint—a “humming, bumming, dinging nasty” noise—all over the upper half of the head, and this part he rubs frequently and nervously with his hand. The noise is in the head, it seems to him, and not in the ears. The patient feels ill and miserable, keeps putting the hand to the head, which he rubs as he stands, and he shakes much and complains of being shaky and miserable ; and again he complains of the noise in the head, which makes him feel as if he would like to destroy himself. He has no melancholic notions of wickedness or uselessness, but there is a deal more than a distinct hypochondriacal tinge in his statement that he is wasting to a skeleton, and has not a bit of flesh on his bones. Hallucinations are denied. There is considerable failure of memory and general dementia—the failing mind of senile decay, precipitated and modified by the results of injury. He denies addiction to drink. He is a thin, wizened, stooping, edentulous ancient ; shaking much, and easily induced so to do, partly from senile tremor of debility ; and, occasionally, when he thinks and complains of it, there is great exacerbation of the coarse shaking, as if half-voluntarily. Sight is impaired, the pupils are slightly sluggish to light ; the gait is slow and slouching, the knee-jerk present.

III.—We now take up the *Third* group of the traumatically-caused diseases of mind in the adult.

In some cases (as *Cases 14* and *15*) severe fracture of skull is followed, after a considerable space of time, by convulsive seizures of epileptiform character ; these increase in frequency, and become associated with the violence after

convulsive seizures and the conditions of mental automatism so often forming part of the manifestation of epileptic mental disorder, and now forming part of those of an epileptoid psychosis. Incoherence, mental confusion, and dementia appear therewith, and increase. Many of the seizures may be quasi-syncopal—a form of epileptiform *petit mal*. Even turning-movements may occur. Local paralysis may come, or tonic spasm, or spasmodic twitches. So may hemiplegia or hemiparesis—partial, or general on the side affected—either only temporarily following the seizures, or else persistent in some degree and temporarily augmenting after the seizures, but ever tending to deepen in degree and, if partial, to extend in range, sometimes with lessened sensibility and lowered temperature on the side affected, between the attacks. The mental state ebbs and flows, fluctuating partly as the seizures appear or hold off, and as it is affected by them; and the patient passes from states in which he is noisy, restless, incoherent, to those in which he is oppressed, languid, or even semi-comatose. There often is some affection of speech, dyslogical—or dysphasic—or dysarthric; and during the post-convulsive pareses or paralyses these speech-conditions are worse. In only some of the cases is this temporary worsening of the speech due to the augmentation of paralysis by convulsive discharge; in most, it is merely an accompaniment of that, and is due to the simultaneous discharge and exhaustion of neighbouring parts of the cortex cerebri devoted to speech-centres and their allies, or due to crippling vaso-motor and vascular disturbances occurring therein. Hence, temporary, or temporarily augmented, motor aphasia, word-deafness, word-blindness, paraphasia, amnesic aphasia, slow speech, pausing speech, hemming speech, syllable-stumbling, quasi-stammering, and blurred, mumbling, and shaky speech. The special senses may become affected, especially is sight apt to fail or cease.

At the necropsy, are changes in the bone at the seat of old fracture, and often osteophyte-like formation of bone-callus on the inner surface; local chronic pachymeningitis; perhaps cohesion of dura, pia, and brain-cortex; wasting, and

variously-produced chronic destructive or indurative changes in the cortex beneath the seat of injury; traces and relics of old sub-dural hæmorrhage, sometimes up to well-formed blood-tumour "cysts;" or of old subarachnoid or pial hæmorrhage, and appearing partly as wasted degenerate areas of the cortex. Counterstroke often has occurred; and, if so, confronting the pole of the cranium, roughly speaking, opposite to the line of incident force, may be the traces and relics of crush or bruise of the cortex, or of meningeal hæmorrhage; and sometimes of sequent local acute and chronic meningitis, in the form of old adhesion bands and meningeal thickening, or old areas of adhesion and decortication, especially about the brain-base.

Atrophy of the cerebral hemisphere chiefly affected has followed, and often is more obvious in the grey cortex than in the subjacent white. Naturally, this atrophy is usually more of the hemisphere on the side the same as the damage to the cranium; but in some cases is more of the opposite side, and, when it is so, is another evidence of counterstroke. The ventricular endyma is often considerably granulated.

In some (much as in *Case 16*) there are marked chronic meningitic thickening and opacities, gradual cerebral atrophy, and perhaps pallor and fine degenerative changes, chiefly in one hemisphere, by far most marked in the frontal and parietal regions, and often irregularly distributed; slight or no adhesion and decortication, granulated ventricles:—gradual failure of mind, sometimes with symptoms, such as self-satisfaction and mild optimism, showing some connecting links with general paralysis.

Nor are these the only cases in which the question of general paralysis arises. For (as an example, *Case 17*) conditions at least resembling, if not, general paralysis occur in some cases;—coming gradually, some months after severe cranial injury, are strangeness of manner, depression, severe cranial pain, hallucinations and delusions; and later on are physical symptoms fairly like those of general paralysis, and mental failure with large ideas—although not very variable—adding to the similitude and

disguise ; if similitude it may be termed, and if we do not admit that traumatic general paralysis (as a lesion) becomes practically cured, for the time being, by vigorous treatment ; although there is final deterioration in mind, speech, and gait. For such patients may live a good many years, and at the late-coming necropsy may present, as morbid naked-eye changes, some slight and chiefly fronto-parietal brain-wasting, some chronic meningeal thickening and opacity, and slightly augmented dural adhesions to the calvaria, together with pigmental and other traces of old sub-dural hæmorrhage. Degeneration of spinal pyramidal tracts co-existing.

These last two sub-groups are closely allied to general paralysis, or from the links between it and some other organic brain-diseases, or, perhaps, are to be taken as modified varieties of general paralysis itself. And there are other cases holding a somewhat analogous position.

Such other cases may have less decidedly the traumatic influence ; as, for example, *Case 18*, with symptoms as if of depressed general paralysis, and indistinct physical signs as in general paralysis. The patient was suicidal, and in some respects dangerous ; the subject of sombre emotional dejection, facile weeping ; delusions and hallucinations of hostility, annoyance, persecution, danger, and hypochondriacal and melancholic delusive notions ; occipital pain since the severe cranial injury four or five years previously, and sometimes frontal pain. The pains were worse at night and increased by warmth. Insomnia. Some symptoms of hystero-neurasthenia. Cardiac disease (left ventricular hypertrophy and dilatation, with leaky valves).

Or, as *Case 19*, with organic brain- and cord-disease from injury to the head five years before admission, and with signs in speech and face considerably resembling those of general paralysis. There were mental depression and weeping ; persecutory delusions of hostility and of condemnation and of impending death at the hands of his torturers, not distinctly systemised ; vivid visual and auditory hallucinations of inimical import, and occasionally leading to excitement. Much cranial pain and tenderness co-existed, as well as spasmodic tremor, increased tendon-reflexes, and an

easily-induced *quasi* terrific shivering or "apyrexial rigor." (For this last symptom, see the writer's "General Paralysis of the Insane," 2nd edit., p. 96).

Another *Case* (No. 20) has links with the next subgroup about to be described. Admitted a week after severe injury to the head and other parts, and treatment at St. George's Hospital, the patient appears to be threatened by traumatic general paralysis; has dementia, moderate loss of memory, and hallucinations; is amblyopic, presents some moderately general-paralysis-like conditions of pupils, tongue, speech, &c. Is in a mentally confused dazed state, which subsequently wears off, and the memory improves; has hallucinations of hearing the voices of the Saviour and of the dead; also, some self-satisfaction, and complaisant, non-chalant acquiescence in asylum life.

Again, there is a group of temporary cases, taking on an appearance of general paralysis, and following quickly or soon after the cranial injury; that is to say, cases simulating general paralysis, but soon clearing up or improving immensely:—

Thus, (*Case* 21) a housewife incurs a severe fall on the back of the head, somewhat to the right side; then, for some weeks, is under treatment in the London Hospital, and an infirmary, whence she enters the asylum under my care; at and about which time she is incoherent, in a confused state, loquaciously recounting a medley of delusional notions, as if expressing dream-experiences; notions of hostility against her, of injury, and of the death of her family, jostling with vague fleeting notions of wealth; and the emotional phases rapidly changing, and expressed in tears or laughter, in quick succession. The physical signs are much as in general paralysis. The diagnosis recorded at the time of admission is that the case is one in which "the signs of general paralysis are simulated by brain-disorder of traumatic origin;" and the patient's recovery lends countenance to the accuracy of the diagnosis.

Another example is *Case* 22, who, about a month before admission to the asylum under my care, falls off a cab and injures his head, when drunk, and is taken to the Great

Northern Hospital, and thence to an infirmary. Injury to the head, an exciting cause, is operative on the basis of drink habit. Physical signs like those of general paralysis, fairly marked. Increased knee-jerk. Impaired memory, sombre feeling, depressive hallucinations, and delusions of hostility and endangered life. But the symptoms clear up much, the memory vastly improves, and now it is found that the patient does not spontaneously remember, and cannot recollect, his former delusions and hallucinations.

From the above sub-groups of cases we pass by easy transition to indubitable general paralysis from injury.

For *General Paralysis from Injury*¹ may occur also, and the cranial or other injury may be a predisposing or exciting factor thereof.

In some examples, at least, of traumatic general paralysis, we find one cerebral hemisphere much more affected than the other by "adhesion and decortication," by a greater extent and degree and duration of the other conditions of the cerebral lesion of general paralysis, including secondary wasting, and sometimes slight or moderate somewhat diffuse induration, and in some a state of lesion partially on the same lines as those of the cases already described at the beginning of this group under the head of convulsions, dementia, &c., following the brain-damage from cranial injury. In some, there are old meningitic thickening and adhesion-bands about the base of the brain and anterior mesial aspect of the cerebral hemispheres; and here cerebro-meningeal adhesion has in some commenced, or here, alone, has occurred; and blindness has often followed the optic-nerve implication in those changes. Secondary descending systematic spinal degenerations are frequent.

In others, the general distribution of the cerebral lesions, and of their respective degrees, in different parts, are of much the more usual type.

Traumatic general paralysis is sometimes marked by the relative predominance or striking nature of such violent

¹ Papers by the writer, *Journal of Mental Science*, Jan., 1883, p. 544; and same *Journal*, October, 1885. And work on "General Paralysis," 2nd edit., pp. 259 and 271.

aggressive symptoms as excitement, noisy raving, violence, destructiveness, in the earlier stages; vivid hallucinations of hearing and sometimes of other, or of all the special senses; often a long-antecedent change of character and disposition, irritability of temper, easily-evoked outbreak of wrath or violence. Here, the brain-lesions and wasting, often chiefly of the right cerebral hemisphere, have been accompanied during life by frequent, recurring apoplectiform and epileptiform seizures, with left hemiplegia, increased knee-jerks, and ankle-cloni.

Or, on the other hand, there are depressed symptoms of hypochondriacal or melancholic type, or delusive notions of hostility, poisoning, persecution, and hence aptness to quarrel or use invective may be unusually prominent.

Thus, in *Case 23* were vivid and horrible hallucinations and illusions of special sense; an irritable, morose ill-temper, the patient, full of hatefulness, cursing, reviling, or threatening for hours together, and monotonously so; right unilateral epileptiform seizures, followed by dextral hemiplegias together with a recurrently more impaired speech; delusional refusal of food; obstinate constipation; a tendency to Raynaud's disease (local asphyxial phase). The adhesion and decortication, and the brain-wasting, of the left cerebral hemisphere predominated over those of the right; the left being three ounces less in weight than the right; at the left posterior central gyrus, postero-parietal lobule, quadrate, and back part of paracentral lobule, was complete separation (when the membranes were peeled off) of the grey cortical substance from the white, and this occurred even at the bottom of the sulci. Heart degenerate. Kidneys, as old changes, capsular adhesions and ordinary cysts; as recent change, slight parenchymatous nephritis.

But expansive symptoms may be, or become, prominent in phases or throughout, and before and with them is the fundamental dementia. Increased tendon-reflexes and striking cloni often attend the later stages; with, or without, hemiparesis, and contractured limbs chiefly on the more paretic side. Pain in the head is, in many cases, severe, especially in the earlier stages. Dangerous, such patients

may be, prone to violence, suicidal, and even homicidal in bursts of excitement. Hallucinatory voices may threaten, or announce danger and ill to, the patient. These marked and protracted prodromes and earlier symptoms may eventually clear off, leaving self-satisfaction—but ever and anon come paralytic seizures of motor helplessness, with mental dulness and oppression, and incapability of writing and speaking, even as well as in the defective way usual to the patient.

Spinal Cord Injury a factor in mental disease.

In my own experience the cases have chiefly been general paralysis and paranoia. But many psychoses originating from spinal injury are psycho-neurotic conditions, built on the foundation of traumatic neurasthenia or hysteria. These last will be taken up presently under the fourth group.

At the moment of cord-concussion or injury the brain may be affected, at least by the propulsion and violent shake of the cerebro-spinal fluid; again, the vaso-motor apparatus of the brain may gradually become affected by the cord's condition: or the morbid process arising from injury may extend from cord to brain.

Case 24.—General Paralysis. Spinal injury, five years before admission, was soon followed by a liability to fits, and by giddiness. Had an early outburst of acute symptoms, with violence, smearing of room with fæces, exalted delusions of possessions and wealth. Later on, ordinary physical signs of general paralysis. Knee-jerk well-marked; fits starting in left hand; slight lead-line on gums, bad taste in mouth (is a painter). Large notions; busy, changing, expansive schemes; ready weeping about trifles.

Case 25.—General paralysis. Spine hurt two years before admission, by an elephant trampling on it. There was some evidence also of sexual excess and syphilis. At admission, were delusions about elephants, and the Emperors of Germany and Morocco, about detectives following the patient, and unseen people talking to him. He made allegations, also, of being abused and ill-treated by his wife and her friends, of being starved, and his home a wreck. Sombre

emotion and dejection. Slight physical signs of general paralysis, affected pupils (reflex iridoparesis, slight myosis), *quasi*-tabetic symptoms. Later on, self-satisfaction; an absurd project of going on the stage as an actor; an impracticable offer of presents to his wife.

*Case 26.*¹—General paralysis. Bad injury to spine, and to head over right parietal bone, six years before recognised insanity. The patient became feeble in health, mentally changed, slovenly, neglectful of duties and orders, depressed, discontented, made silly unfounded charges against those about him. Later on, he was restless, excited, irritable, noisy, mischievous, destructive to clothing, sleepless, peevish, sulky, dull, with outbursts of rage; gave absurd and confused orders; imagined and said he had placed others under arrest. Under my care, he had exalted delusions, and absurd delusions of ill-treatment, of starvation, of daily frightful corporeal injuries inflicted on him, of his life being threatened, and attempts made to poison him. Though at times buoyant, he was usually sullen and morose; often threatening and full of invective. There were hallucinations of sight and hearing; and muscular illusions as to the flight of his body. Once, temporary left hemiparesis, and once left facial paresis.

Case 27.—Paranoia. Some years ago incurred an injury to the spine by a fall, and has been affected by this, off and on, ever since. Has auditory, tactile, and thermic hallucinations and paræsthesiæ; delusions of being conspired against and persecuted by personators, who blow on him with blow-pipes in order to make a living out of him thereby, and who jeer and scoff at him, and conspire against him to benefit themselves. The gait is constrained and embarrassed; at every step one whole side of the frame is moved stiffly, and thus each side alternately.

Originating in spinal injury are some cases of the next group, also.

We resume the subject of the chief groups of mental disorder in *adult* life with *cranial* injury a factor.

IV.—The *Fourth* great group of mental disorders follow-

¹ Reported by present writer, *Journ. Mental Science*, Jan., 1883, p. 548.

ing injury consists of functional cases of a special type, and more frequently seen by the neurologist and surgeon than by the mental physician. In the more simple examples, indeed, the symptoms are predominantly "nervous" (in the restricted sense) rather than psychic. But in all cases an element of psychic perversion or failure is present; and in the more severe cases it takes the leading rôle in the morbid drama. Similarly-produced cases with definite organic cerebro-spinal lesions and symptoms find place in other of these groups, notwithstanding that they, also, are often attended by symptoms of traumatic neurosis, neurasthenia, or hysteria, described below.

In this fourth group, thus limited, come many cases following cerebral or cerebro-spinal shock; many examples of the traumatic neurosis which is engendered with especial facility should there happen to be a pre-existent neurasthenic or hysteric basis, but which may be produced independently of the pre-existence of these; and which, on the other hand, may become manifest as, or may occasion, traumatic neurasthenia, and subsequently on this basis, traumatic hysteria ("hystero-neurasthenia"), the building-up of a traumatic psycho-neurosis; for the mental elements are so striking that the state is as much a psychosis as a neurosis. Consequently, here come a considerable number of the examples of so-called "railway brain" or "railway spine," "spinal concussion;" "functional," "ideal," "psychical," paralyzes, &c., accompanied by psychic change, immediately occasioned by injury, which perhaps may be slight. In such cases the mental and cerebral conditions are supposed to be somewhat analogous to those of the somnambulic phase of major hypnotism, in which similar motor, sensory and sensorial anomalies are easily produced by suggestion. For it is supposed that, after the injury, there is a cerebral state in which "suggestibility" is increased, and that the "traumatic suggestion" arises from the "traumatic shock" of the injury; the state of "general nervous commotion," or of "nervous shock" implying, or determining and securing, a cerebral condition and mental modification in which such "traumatic suggestion" easily arises; so that, mental spon-

taneity, will and judgment being obscured, the ideas of sensory and motor failure become realised, the temporary sub-sensitive and paretic states of the limbs due to, and forming the clinical phenomena of, "local shock" (which are hypæsthesia and paresis, even in the healthy) becoming—perhaps by the co-existent emotional perturbation—enhanced and transformed into complete paralysis, &c., initiated by a process of "auto-suggestion," and developed by a process of mental elaboration, in the unstable neurasthenic hypochondriacal anxious self-contemplating—or hysterical—condition of the mind of the subject of traumatic neurosis. And we find neurasthenia and hysteria in many cases to be the basis on which an injury, perhaps slight, leads, by those processes of mental elaboration and auto-suggestion, to grave symptoms, and eventually to a state of traumatic neurosis and traumatic psychosis.

The term concussion of the brain or cord seems to me to be capable of use, and to have been confusedly used, in three different senses:—

(1) As the manner of receipt of the injury (concussion as a cause, or in an ætiological sense).

(2) As the pathological condition of the organ, the primary, impalpable, undemonstrable injury thereof, inflicted by the results of the blow (concussion as a pathological effect).

(3) As the symptom-complex arising from ("2") that last-mentioned pathological condition of the organ (concussion as a clinical phenomenon).

Of these, the second seems to me to be the important one, and is the one I mean when speaking of concussion, unless it is otherwise specified. "Shock," or "nervous shock," may be used as depression or exhaustion of the cerebro-spinal nervous system by some sudden impression or violence.

The chief symptoms of the hysteroneurasthenic group are intellectual perversion, emotional dejection, sombre sad ideas, failing power of volition, petulant irritability or rage, headache, insomnia, sexual weakness, spermatorrhœa, and sacral backache.

With hysteric hemianæsthesia usually go contracted visual field and achromatopsia. On the other hand, severe bladder-troubles, reflex irido-plegia, and optic nerve atrophy point to organic changes.

When the physical cerebral or cerebro-spinal shock from external violence has been more considerable—after the symptoms of the primary shock (namely, bewilderment, mental confusion, and perhaps a quasi-somnambulic automatic state) have passed off; then, when consciousness returns, come those symptoms which are preliminary to the traumatic neurosis or overt psychosis—viz., loss of memory of the life between the moment of accident and the return of full consciousness (after the period of defective consciousness a moment ago described), and sometimes retrospective loss of memory of a space of time immediately preceding the injury; also back pains from sacrum to nape; headache; malaise; uneasy disquietude: insomnia.

Next come the symptoms of the overt psychosis—melancholia, often of hypochondriacal type, with great irritability. Sadness, dejection, indifference to friends and family, distress, oppression, sombre feeling, rising into fear, and culminating, perhaps, in seizures of terror or in suicidal attempts; despair (often with præcordial pain, oppression and palpitation). Agitated, variable, fickle and tumultuous emotional changes; vivid and terrifying recollections of the accident or injury, terrifying dreams anent the same. The patients are easily agitated, easily mortified, abnormally sensitive to impressions from without—*e.g.*, they are perhaps easily thrown into emotional and physically-expressed tumult by ordinary noises. Frequent, are self-study as to symptoms and concentration of mind upon them, irritable irascibility, anxiety, inclination to delusive notions as to being annoyed; vague, torpid, confused, easily fatigued mental operations, hence a careless slipshod way of asking or answering questions; failing general memory, or lacunar memory; failing and easily fatigued attention, rapidly-coming confusion in reading; silence, slow replies, volitional limitation, hallucinations, insomnia; severe loss of memory, possibly even of patient's own name.

Irregular and intermingling anæsthesia and hyperæsthesia of cutaneous, or of general, sensibility, or of special senses, or of all these, and often of hysterical hemianæsthetic or hypæsthetic character, in zones or segments and not following the course and distribution of nerves; sometimes impaired muscular sense, analgesia, contracted visual fields, a- and dys-chromatopsia, cloudy vision from paresis of accommodation, impaired hearing, dull heavy back-pain, constrictive head-pain—increased by intellectual effort. The pains are usually heavy, obtuse, and worse on movement. Sparkles, flying spots, or colour-play, before the eyes; noises in the ears, hyperacusis; morbid tastes and smells; spinal tenderness; paræsthesia—*e.g.*, morbid sensation of heat or cold or formication.

Defective motor power, irregularly distributed; paralysis, perhaps of limbs on same side as injury. If hemiplegia is present, the face and tongue are not affected, nor is palsy of any cranial nerve at all frequent; it often is more marked in the leg than in the arm, and often with it is partial or unilateral anæsthesia; or there may be palsy of a segment of a limb only. Usually the paralysis is flaccid. The muscles may be wasted, and their electric excitability be quantitatively diminished. The gait may become timid, slow; with short steps, feet wide apart, stiff trunk, frequent halts, and often much tremor. Tendon reflexes usually increased. Tremor, spasm, vertigo. Perhaps sluggish unequal pupils. Speech slightly slow, explosive, interrupted; or, in some cases, ceasing temporarily from forgetfulness.

Sometimes vaso-motor rapid changes, and heightened cardiac impressibility, *e.g.*, attacks of palpitation, quick pulse, flushed face, dilated pupils; or, attacks of sweating of the axillæ, with rapid pulse, reddened face and chest; and blue tint of the palsied parts.

Anorexia, disordered digestion, failing nutrition, failing virile power, polyuria, glycosuria.

Thus, in the foregoing paper, I have described four great groups of cases in the *adult*, with *cranial* injury a distinct traumatic factor in mental disease:—the first con-

sisting of ordinary forms of functional psycho-neuroses; such as a form of mental automatism, stuporous insanity, acute and subacute hallucinatory insanity, and melancholic depressed states; the second comprising paranoia and its immediate congeners; the third group constituted of traumatically-caused severe organic brain-disease, with dementia, or other mental conditions, and, often, convulsions, paralyzes, or other motor, and various sensory and sensorial, phenomena; the lesions being either local gross destructive ones, or those of chronic cerebritis, or sclerosis, or of cerebral atrophy—perhaps chiefly unilateral—or those of general paralysis of the insane; and the fourth group consisting of functional cases of a particular type.

Partly in addition to the numbered cases, the clinical and necroscopical descriptions and summaries relating to the first and third of the above great groups are entirely drawn from my cases; those relating to the second group are almost entirely so; whilst, as regards the fourth group, I have utilized cases under my care, and cases under the care of others, as well as the descriptions of Erichsen, Herbert Page, Charcot, Oppenheim, Clevenger, and Blocq.

The numbered cases were all under my care, and I have purposely limited the descriptions of them to very concise summaries of the principal clinical or necroscopical features.

CEREBRO-SPINAL MENINGITIS.

(*With a record of thirteen cases.*)

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THE thirteen cases of cerebro-spinal meningitis, of which details are given here, illustrate some of the important relations of this disease. It would certainly appear that this form of meningitis occurs more frequently than is usually supposed, and there are several reasons why it should be overlooked. In the first place, it is occasionally so rapidly fatal (case 4) that there is hardly time to make a diagnosis. If the disease occur in a child, the difficulty may be even greater; so that Lewis Smith, basing his statistics on cases occurring in New York, says ("Cyclopædia of Children's Diseases," vol. i.): "My observations lead me to think that the younger the child the more frequently is cerebro-spinal meningitis overlooked and some other disease diagnosed." Moreover, it is recognised that children are more susceptible to this disease than others. Again, if it occur in the course of acute disease, its symptoms may be so masked that it is almost impossible to recognise it. Not only is this disease apt to be overlooked during life—it may escape notice even in the *post-mortem* room. In one case it was difficult to say whether the membranes of the brain were really diseased or not. An examination of the spinal cord, however, revealed the nature of the case—most obscure during life, for it was an example of the 'foudroyant' disease. Schultze of Heidelberg (*Berl. klin. Wochenschr.*, 1887) particularly pointed out that characteristic naked-eye appearances must not always be expected. He instances the case of a patient dead of meningitis after a three weeks' illness, in which only slight changes were found in the membranes, whereas a minute

examination of the brain showed the round-celled infiltration in the cortex, so often present in this disease. An interesting case is referred to in the *Brit. Med. Journal* of 1887, in which a sacral tumour had been removed Erysipelas supervened, and the symptoms present were those of a cerebro-spinal meningitis. At the necropsy the membranes of the cord were healthy to the naked eye, but the fluid examined by M. Tavel showed, by cultivation and otherwise, abundant streptococci. The author says that therefore the diagnosis of meningitis was correct. It does not follow that the spinal meninges are not involved because there is no exudation about the medulla and upper cord. Most often the exudation is much more plentiful in the dorsal and lumbar regions. The disease is really one of the subarachnoid tissue, involving on the one hand the pia mater and subjacent nervous tissue, and on the other the arachnoid. The reason that the exudation is more abundant in the dorsal and lumbar regions of the cord is, it would appear, because the subarachnoid tissue is more plentiful there. The same cause, and not gravitation, explains the fact that the exudation is chiefly, if not exclusively, found on the posterior aspect of the cord. In some observations by Dr. Carrington (*Path. Soc. Tr.*, 1882 and 1884) purulent lymph was present on the anterior surface, and in one case nowhere else. This experience would appear to be exceptional—different from most of the recorded cases where the cord has been examined and the distribution of the lesion specified. In one of their cases, Neumann and Schäffer (*Virch. Arch.*, 1887) state that there was pus between the pia mater and the cord, but more usually the pia mater is adherent to the parts beneath. The disease is really a meningo-encephalitis and myelitis, as Strümpell (*Deut. Arch. f. klin. Med.*, 1882) long ago pointed out. This spreading of the inflammation inwards, and not the mere pressure of fluid in the ventricles, is chiefly responsible for the coma.

The question as to how far the morbid changes may go and yet be consistent with recovery, is an interesting one. Popoff (*Berl. klin. Wochenschr.*, 1886), analysing three cases of which two were fatal, says of the third that the exuda-

tion must have been serous or sero-fibrinous. But when two patients are observed side by side, both apparently moribund, and the one recovers whilst the other dies, it is difficult to imagine to oneself that the morbid changes are very different in these two cases, provided the disease has lasted a certain time. And yet in the fatal case much purulent exudation is found at the necropsy. Dr. Hadden's case (*Path. Soc. Tr.*, 1885) is one of great interest. The disease had lasted over fifteen months, the patient ultimately dying of it. The under, and to a lesser extent the upper, surface of the cerebellum, the adjacent parts of the cerebral hemispheres, as well as the posterior surface of the cord, were covered with a layer of white opaque material. The arachnoid was mostly free, but was in parts adherent to this subjacent layer. There was thickening and opacity of the membranes about the optic tracts and chiasma. Strümpell (*loc. cit.*) reports the case of a patient who died in three months' time. He says that there was a chronic, but in no place purulent, meningitis. Dr. Bristowe's case (BRAIN, 1888-9) lasted some seven weeks; soft lymph was found on the under surface of the pons, about the medulla and cerebellum; there were only slight traces of inflammation in the spinal membranes. It is not impossible that, with a more systematic examination of the cord, there might be found at times evidences of past inflammation which at present escape our notice.

Cerebro-spinal meningitis is said to be (1) primary when it occurs in its epidemic or so-called 'sporadic' form; (2) secondary when it complicates some acute disease, like pneumonia, enteric fever, &c.; (3) consecutive when it follows upon bone affections due to ear or nose disease, and (4) traumatic. The following are examples of the so-called sporadic disease. (It may be stated once for all that with the exception of Nos. 3 and 12, there was no evidence either before or after death of ear disease in the thirteen cases reported here and that no tubercle was found anywhere in the twelve necropsies):

CASE I.—Norwegian lad, æt. 19. Severe headache for four days. No vomiting. On the sixth day drowsiness and pain in

the head, which latter is much retracted. Herpes at right angle of mouth. Temperature 102. Abdomen retracted. No optic neuritis. Slight twitchings about mouth in the evening. Seventh day: delirium, hydrocephalic cry, general hyperæsthesia. Eighth day: semi-coma. Herpes now symmetrical. Ninth day: patient is more comatose, swallowing difficult. Temperature 108 shortly before death, 109.6 shortly after; *P.-M.*, pus at base of brain and over hemispheres in places, also over posterior surface of cord in lumbar and dorsal regions and about medulla. Lungs œdematous. No tubercle anywhere. (May, 1888.)

CASE II.—Girl, æt. 3 $\frac{3}{4}$. Three other children in the same family dead of convulsions before twelve months' old. Sudden onset with vomiting. Fits on second day; admitted comatose. Convulsions frequent, more limited to one side. No optic neuritis. Third day: comatose; twitching almost limited to one side of face. Death. *P.-M.*, suppurative meningitis both cerebral and spinal. (Sept., 1889.)

CASE III.—Girl, æt. 15. First day: headache, severe and repeated vomiting. Fifth day (when admitted): semi-comatose, unable to swallow fluids, herpes, neck rigid and head retracted. Sixth day: unconscious, retention of urine. Patient then improved a little, but had a relapse on the eleventh day. On the thirteenth day she was conscious but very deaf; fourteenth day there was pus in the urine; twentieth day, vomiting again; twenty-first day, two rigors, very wakeful; twenty-seventh day, relapse was well marked, severe vomiting, &c. From the forty-second day onwards there was steady improvement. During convalescence the gait was staggering; this ultimately passed off, but the deafness remained. There never was any otorrhœa. (July, 1890.)

CASE IV.—Man, æt. 59. Woke up in the morning screaming, later unconscious. On admission he was noisy though unconscious. Right pupil dilated. Violent convulsions. Albumen in the urine, coma, and death within twenty-four hours of onset. *P.-M.*, some opacity of the membranes of brain, pus in the spinal meninges. (Sept., 1890.)

CASE V.—Girl, æt. 8 $\frac{3}{4}$. Onset sudden, with headache and vomiting. Drowsiness. Eighth day: convulsions which continued up to time of death, affecting chiefly the left side. Death on fourteenth day. No *P.-M.* Nature of meningitis open to some doubt. (Sept., 1890.)

CASE VI.—Woman, æt. 30. Seven months pregnant. Headache for a few days, then vomiting and frequent convulsions.

Death. *P.-M.*, a diffuse suppurative meningitis. Fœtus and uterus healthy. (Aug., 1890.)

That cerebro-spinal meningitis occurs as a complication of acute pneumonia has long been recognised. Dr. Wilks described such a case in the *Lancet* of 1865. Immermann and Heller emphasised the relation between these two diseases in 1868. In a series of cases of meningitis reported by Dr. Greenfield (*St. Thos. Hosp. Rep.*, 1877) pneumonia was present in three. In the *Deut. Archiv. f. klin. Med.*, 1881, Nauwerk related some seventeen cases of cerebro-spinal meningitis complicating acute pneumonia, and he advanced an embolic theory to account for the disease. The frequency with which it occurs, varies very considerably as shewn by the figures given by Netter (*Arch. gén. de Méd.*, 1887). During or after an epidemic, as Lewis Smith points out (*loc. cit.*), this disease may occur as a complication of other acute affections with unusual frequency. Netter (*loc. cit.*) set himself to prove that meningitis might occur in the course of acute pneumonia (as in cases 7 and 8), that it might precede or follow it (as in case 10), or occur without it. Like cerebro-spinal meningitis acute pneumonia may unquestionably occur in epidemics. Cerebro-spinal meningitis may apparently be communicated from one individual to another (see Kohlmann, *Berl. klin. Woch.*, 1889); and so can pneumonia, as may be illustrated by five cases occurring in the same household (Kempff, *Internat. Journal of Med. Sci.*, 1887); or by the cases arising in the ward of a hospital, as reported by MM. Lancereaux and Besançon (*Arch. gén. de Méd.*, Sept., 1886). Further epidemics of cerebro-spinal meningitis and acute pneumonia may go hand in hand, as in the instance reported by Runeberg (*Berl. klin. Woch.*, 1888).

Two epidemics, investigated by Dr. Bruce Low ("Report to Local Gov. Board, 1891), may be referred to here, and I am indebted to Dr. Low for many details respecting them. The first epidemic (of pneumonia) occurred in Raunds in Northamptonshire. Ninety-three households, representing one hundred and fifty-five people were attacked. Young subjects were mostly affected. In some, meningeal symptoms were noted, and in some, discharge from one or both ears. Tonsillitis

was prevalent. In one case (boy æt. 13) there were first signs of acute pneumonia, later of meningitis, and the patient died. There was reason to believe that the disease could be communicated from one individual to another. The second epidemic, in Heyford, $6\frac{1}{2}$ miles from Raunds, was very similar, but meningeal symptoms (with retraction of the head) were more marked. Of two fatal cases, Dr. Fuller noted purpuric spots in one. One case, in a child, æt. 18 months, was a typical example of cerebro-spinal meningitis with recovery. Space prevents my dealing at greater length with these reports, as well as with another, also by Dr. Bruce Low, in which an epidemic of cerebro-spinal meningitis in the Eastern Counties, in 1890, is detailed. They are of great interest, and point to the close relation of cerebro-spinal meningitis and acute pneumonia. Of course it must be remembered that pneumonia in children may be accompanied by brain symptoms—cerebral pneumonia. Maurier, as far back as 1874, dwelt upon this (*Deut. Archiv. f. Klin. Med.*). Indeed, the parallelism between acute pneumonia and cerebro-spinal meningitis is at once striking and extraordinary; so that Runeberg (*loc. cit.*) says that meningitis is one of the many localisations of the pneumonic poison.

The following are two examples of acute pneumonia, complicated by cerebro-spinal meningitis:—

CASE VII.—Man, æt. 30. Acute pneumonia (Rt.). Very little delirium during the attack. No crisis. On 14th day, stupor. On 15th, comatose, after a very delirious night. Temp. 105. Death. P.-M., Rt. lung completely solid. Much pus at the base of brain and about medulla, a little here and there over convexity. In the spinal cord much pus over the posterior part in the lumbar region. (June, 1888.)

CASE VIII.—A woman, æt. 30. Acute pneumonia (right base). A little delirium during the attack. On the 9th day in the afternoon, she was apparently dosing quietly. Later, she could hardly be roused. Swallowing was difficult. The coma gradually deepened. Temp. 104.8 shortly before death. P.-M., Membranes of the brain somewhat opaque, but changes not very marked. In the spinal meninges there was pus. (Feb., 1890.)

Malignant, or infective endocarditis, also stands in a special relation to cerebro-spinal meningitis (and also to acute pneumonia). Among Nauwerk's cases (*loc. cit.*) there were two with fresh endocarditis. Osler (*Gulst. Lect. Brit. Med. Jour.*, 1885) said, that out of one hundred and three autopsies on acute pneumonia, malignant endocarditis was present in eleven, and in five of these eleven cases there was meningitis. Among fourteen cases of ulcerative endocarditis, reported by Byrom Bramwell (*Internat. Jour. of Med. Sci.*, 1886), there was croupous pneumonia, at least in two, and probably in seven. It has been pointed out by Osler (*loc. cit.*), Jaccoud (*L'union Med.*, 1889), Sée (*Bulletin Med.*, 1888), and others, that in cases of so-called ulcerated endo-carditis, there is not always a loss of tissue, but at times even a proliferation (proliferative or vegetative endocarditis). M. Sée says that this vegetative form is due to the pneumococcus, and is less virulent than the ulcerative form arising from infection with the streptococcus.

The following is an instance of malignant endocarditis with meningitis:—

CASE IX.—Man, æt 32, in hospital for mitral and aortic disease. He first had intense headache and pain in his neck. Then vomiting, intermittent temperature, and stupor. Cheyne-Stokes breathing. Hæmaturia. Up to his death on the fourth day, he would wake up at times complaining of his head. *P.-M.*, Purulent exudation in meninges over hemispheres, also at base of brain and round about medulla. Recent vegetations on aortic valves (two cusps fenestrated). Old disease of mitral valve with new vegetations spreading into auricle. Kidneys presented appearances of acute Bright's disease. No infarcts. Condition of base of right lung doubtful. (Jan., 1891.)

The following is an example of all these three diseases occurring together:

CASE X.—Man, æt. 30. Acute pneumonia (left base), herpes on the forehead, face and lips. During apparent convalescence temperature rose one evening. The next day he had headache and was restless. Vomiting. Signs of consolidation over right apex. Sputum blood-stained. Delirium, coma and death. *P.-M.*, left lung nearly recovered. Right apex solid. Under surface of one aortic cusp covered with a mass of recent vegeta-

tions (proliferative endocarditis). Patches of suppurative meningitis over cerebral hemispheres. Spinal meninges apparently healthy. (May, 1889.)

As these three diseases have been known not infrequently to occur together, there would appear to be some *primâ facie* reason to suspect that the materies morbi causing the one might, under slightly altered circumstances, produce the other; or, in other words, that in an individual some micro-organism will bring about under one set of conditions an inflammation of the lung, under another an acute endocarditis, under a third a cerebro-spinal meningitis, and under others any possible combination of these diseases. These altered circumstances are made *in experiments on animals* if the valves are damaged after Rosenbach's method, or the meninges injured as was done by Netter and others; and they are seen *clinically* when the disease attacks a valve already damaged by a previous endocarditis, or selects by preference the meninges of the alcoholic. As unfortunately no experiments of any kind could be undertaken in the cases reported in this paper, suffice it to say that modern bacteriological research has amply explained the clinical association of these diseases. Among the micro-organisms which have been proved capable of setting up a cerebro-spinal meningitis, the pneumococcus (of Fraenkel) may be instanced as having been found, bacteriologically as well as morphologically, in the exudation in the meninges, in the vegetations on the valves, and in the pneumonic consolidation.

Cerebro-spinal meningitis may complicate diphtheria or perhaps a simple tonsillitis. The following is an instance of its complicating diphtheria.

CASE XI.—Man, æt. 68. Sore throat, on the third day he was found comatose with a right hemiplegia and conjugate deviation of the eyes to the left. Death. *P.-M.*, greenish-yellow exudation in the cerebral meninges. Purulent infiltration in spinal meninges. There was membrane present in the larynx. (June, 1890. Dr. Churton will publish this very interesting case in full.)

Influenza is sometimes followed by cerebro-spinal meningitis. The following notes of a case which occurred in

Leeds during the recent epidemic were made by a very competent observer :

A child, æt. 5, had influenza followed by apparently croupous pneumonia, as shown by the physical signs and rusty sputum. The case did not clear up, and at the end of the fifth week of the pneumonia the child was seized with vomiting, convulsions and then coma. It looked moribund. Evacuations were passed under it. It soon improved a little, its temperature being sub-normal for several days. Four weeks after the commencement of the meningitis (marked by the vomiting, &c.) the child relapsed and died in convulsions. No autopsy.

The combination of influenza, acute pneumonia, and meningitis, is strongly suggestive that the meningitis is not tubercular in nature. If in addition, the course of the meningitis were unlike that of tubercular meningitis, however anomalous, the evidence becomes stronger. This was the case here. Last year, some interesting cases were re-recorded in the *Brit. Med. Journal*, by Dr. Bristowe, and also one by Dr. Nicholson, in connection with influenza. The relation of cerebro-spinal meningitis to enteric fever has also been pointed out, and among others, by Steiner (*Deut. med. Woch.*, 1887). An interesting case was reported by Roux (*Lyon Médical*, 1889). Wolff has shown, however, (*Deut. Archiv. f. klin. Med.*, 1888), that in enteric fever, meningeal symptoms may be present during life, and yet no meningitis be found after death. The tubercle bacillus rarely gives rise to suppurative meningitis, though it should be borne in mind that tubercular meningitis affects the spinal, as well as the cerebral meninges. Wolff says (*Deut. med. Woch.*, 1887) that where a purulent exudation exists, the proof may be wanting as to whether it is really tubercular. In a case of suppurative meningitis, reported by Ortmann (*Archiv. f. exper. Path.*, 1888) there were grey and yellow tubercles in the apices of the lungs, but none in the meninges or other organs. There were no tubercle bacilli in the pus from the meninges, whereas the lanceolate pneumococcus was found in it, and experimented with.

Cerebro-spinal meningitis has occurred in connection with measles, as shown by the cases of Dr. Goodhart (*Clin. Soc. Tr.*, 1886), and Ulrich and Smith (Virchow and Hirsch's

Jahresbericht, 1887). It has also been known to complicate erysipelas.

The relation of ear disease to cerebro-spinal meningitis is one of particular interest. Disease of the petrous bone may set up a meningitis, and this meningitis extend down the cord. This class of case, in point of origin, is more or less clear; but there is another group, of which the same cannot be said. Here the otorrhœa has, perhaps, lasted a long time, and at the necropsy no bone disease is found. It has been suggested that the pus in the middle ear forms a good cultivation ground for micro-organisms—such as the pneumococcus—whence the infection of the meninges. It is interesting to compare this with the very suggestive paper referred to above, in which Dr. Goodhart said in 1886, when speaking of cerebro-spinal meningitis, and zymotic disease: "That the otitis interna, *i.e.*, media, or whatever it may mean, forms a fructifying ground, by which the adjacent membranes become attacked; that it forms the open door by which the murderer steals in, rather than the weapon by which the injury is inflicted; that though the otitis interna is at the bottom of these cases, the acute inflammation may still own a septic or zymotic origin."

Now, there can be no doubt that a suppurative catarrh of the middle ear may arise in the course of a cerebro-spinal meningitis, and the following case apparently illustrates this point:—

CASE XII.—Patient æt. 16. Confined six weeks previously. Was up for a week, fourteen days after confinement. On admission she complained of weakness in the legs and deafness. The pupils were dilated. Three days later, profuse otorrhœa (Rt.), vomiting, and later coma. Twitching of the right arm and face were noted before death. *P.-M.*, most abundant pus over the hemispheres of the brain, at the base, and also over posterior part of cord in the dorsal and lumbar regions. Recent lymph on both pleuræ. Small pyo-salpinx (old). Inside of uterus healthy. No disease of petrous bones. (March, 1891.)

In the *Lancet* (1900, vol. ii., p. 918) Dr. Larsen relates a case in which muco-pus was found in the middle ear and in the mastoid cells, and a reddish pulpy tissue in the semi-circular canals. There could be no doubt that the disease

arose in the course of the meningitis. It spread from the meninges through the labyrinth into the middle ear. In another case which Dr. Larsen refers to, changes even more advanced were found in the labyrinth, but these had not extended any further outwards. This may be the explanation of the deafness not infrequently present after meningitis (case 3). In an article on meningitis and otitis interna in 1872 (*St. Barth. Rep.*), Dr. Gee described three groups of cases: (1) The symptoms of otitis precede those of meningitis; (2) the symptoms of meningitis precede those of otitis; (3) the symptoms of otitis are latent throughout. This authority, however, was inclined to think the otitis was the cause of the meningitis in all these instances. Aural surgeons, and among them Dr. Barr, bear witness to the fact of how often children are seen with bone deafness, the origin of which lay in some acute illness of an obscure nature. Very probably this may have been cerebro-spinal meningitis. If so, it affords additional evidence to the greater frequency of the disease than is generally believed. A case of Weichselbaum's (*Virchow and Hirsch's Jahresbericht*, 1889) may here be referred to in which there was a general infection with the bacillus of pneumonia (Friedländer's) starting from an acute otitis media. The patient had an acute rhinitis, a phlegmon of the sterno-mastoid muscle, and a commencing pneumonia of the left upper lobe. The above-named bacillus was cultivated from all these lesions.

There is a class of traumatic cases of exceptional interest. During the epidemic of pneumonia in Leeds in 1889 a man, æt. 45, received a slight injury to his head and chin. The next day, when walking out, he had a fit. The following day he was hemiplegic and drowsy, and the day afterwards he died. A suppurative meningitis was found, and two inconsiderable bruises on the head, but no fracture. Homen and Saltzmann reported just such a case of cerebro-spinal meningitis occurring after a fall on the head but with no fracture (*Fortschr. der Med.*, 1890, No. 19).

Cerebro-spinal meningitis has been known to occur after

operations (mostly trivial) on the nose and throat, and the following case illustrates it :

CASE XIII.—A woman, æt. 70, had her right tonsil removed for sarcoma. She was up, about and well after the operation. Five days later she woke up moaning, pulse irregular. Temp. 100.6. She was very restless, but could not speak. She soon became unconscious. On the second day much the same. On the third day she was rather better, conscious and able to speak a little, but became worse again in the evening. She died comatose on the fourth day. *P.-M.*, there was pus over the convexity of brain, as well as at the base, and also over the posterior part of the cord in the lumbar and dorsal regions, but none in the cervical. Operation wound healthy. (Sept., 1889.)

I have to thank Mr. Lennox Browne for the details of one such case and a reference to another. The nature of the connection, if any exist, between the operation and the disease is doubtful. Pneumococci are unquestionably present in the mouth and nose (Fraenkel, Netter, &c.) and more abundantly in the case of those who have suffered from acute pneumonia.

On the other hand, cerebro-spinal meningitis not infrequently begins with a catarrh of the nose or throat (Strümpell, Weigert). The prevalence of tonsillitis in the above named epidemics, reported by Dr. Bruce Low, is interesting in this respect. In individuals whose membranes are for any reason more vulnerable, it is not improbable that micro-organisms, finding access at the wounded surface, develop and thus bring about the disease. Ortmann (*loc. cit.*) says : “It seems plausible to explain a meningitis which supervenes on a lesion of the respiratory or alimentary tract by the penetration of the pneumococci by way of that lesion,” and Senger (*Arch. f. exper. Path.*, 1885), “That every wound of the skull and every otitis media which makes, so to speak, a connection between the outside world and the cranial cavity may give occasion to an artificial yet natural inoculation.”

Cases 2, 4, 8, 9, 10, 11 and 13 occurred in the Leeds General Infirmary; Nos. 2, 8, 9 and 10 being under Dr. Eddison, No. 4 under Dr. Barrs, No. 11 under Dr. Churton, and No. 13 under Mr. Jessop. Cases 3 and 12 occurred in the Leeds Fever Hospital under Mr. Pearson, and Case 6 in

the Leeds Union Infirmary under Dr. Allan. Cases 1 and 7 (published in the *Lancet*, 1888) occurred in the Seamen's Hospital, Greenwich, under Dr. Anderson and Dr. Curnow respectively. I am much indebted to these gentlemen for permission to use the cases.

HEMISECTION OF THE SPINAL CORD :

AN EXPLANATORY NOTE.

BY WILLIAM ALDREN TURNER.

As it has been represented to me, since the publication of my paper on "Hemisection of the Spinal Cord," in the Winter Number of this Journal (vol. xiv., p. 496), that I did not refer to a paper on a similar subject by Dr. F. W. Mott in the *Proceedings of the Royal Society* or to certain statements that were made at a meeting of the Neurological Society in June, 1891, when three monkeys on which hemisection of the spinal cord had been performed were exhibited, I am prompted to write the following explanation :—

The research, the results of which are recorded in my paper, was suggested after perusal of the abstract of a paper by Dr. F. W. Mott published in the "Proceedings of the Physiological Society," in the *Journal of Physiology*, January, 1891. In that paper Dr. Mott controverted certain of the views regarding the paths of sensation in the spinal cord which had been more or less accepted by physiologists and clinical observers since the publication of the well-known researches of Dr. Brown-Séquard. It appeared to me, therefore, as facts had been advanced by Dr. Mott in opposition to Brown-Séquard, that the subject required re-investigation, and Dr. Ferrier kindly put his laboratory and the necessary material for a research at my disposal. The experiments and observations which I recorded were commenced on April 11th, 1891, and were conducted at intervals almost up to the end of that year. On June 10th, 1891, three of the animals, on which experiments had been performed, were exhibited by Dr. Ferrier and myself at a meeting of the Neurological Society. A short paper was read by me at the same meeting, and the conclusions, which I believed to be warranted, were stated. In the course of the discussion Prof. Schäfer and Dr. Mott took excep-

tion to certain of the observations which we had made.¹ I did not consider it necessary to state their objections in my paper in BRAIN, as I could not hold myself responsible for the observations and opinions of others, but thought it better to leave it to themselves to publish their criticism in a form which they might think suitable.

On June 18th, 1891, Prof. Schäfer communicated to the Royal Society a paper by Dr. Mott, entitled "Results of Hemisection of the Spinal Cord in Monkeys." At the time when my paper was written I did not know that an abstract of this communication had appeared in No. 302, p. 120, of the *Proceedings of the Royal Society*, issued on August 28th, 1891, and it was not until nearly the end of January of this year that I was made aware of its publication. I have since read the abstract, and can now state that the points of interest summarised in it correspond very closely with those given in Dr. Mott's communication to the *Journal of Physiology*, to which reference has already been made. I, therefore, feel that the perusal of that communication had, so far as is possible in a *résumé*, put me into the position of ascertaining in what respects his conclusions differed from those formulated by Brown-Séquard. I should mention that I was present at the meeting of the Royal Society when Dr. Mott's paper was communicated, and that I received a print marked "uncorrected proof exclusively for the use of the Fellows at the Meeting of the Society," containing an abstract of it. In preparing my paper for the press I was unaware that I was entitled to quote or even refer to a proof so marked. Had I known that the abstract had been published in the above number of the *Proceedings*, or that I could have made public reference to the "uncorrected proof," I should certainly have done so, as it was far from my intention to ignore or neglect Dr. Mott's researches in this department of neurology, or to show any want of courtesy to him.

¹ Dr. Mott, to whom I have submitted this explanation, desires me to state that he and Prof. Schäfer, having previously tested two of the animals, denied the existence of anæsthesia on the non-paralysed side. The third animal was not tested.

Clinical Cases.

CASE OF EXOPHTHALMOS, FOLLOWING EPILEPSY.

BY J. B. NIAS, M.B. OXON., M.R.C.P.

Physician to the Western General Dispensary.

H. S., labourer, aged 33, on August 8th last, while working in a house went down into the kitchen about breakfast time to get some water. While there suddenly fell down unconscious, and was found so by his companions; is said to have struggled and moaned while in the fit, bit his tongue, and blackened his eye in falling. Recovered in $1\frac{1}{2}$ hours, and was able to walk to the Western General Dispensary, where he was prescribed for and sent home. Continued after this at work, but suffered from a continual headache on the left side of the head. On August 18th while at work was seized with great giddiness and confusion of intellect, but did not fall or lose his senses. On August 22nd, having obtained a patient's letter, came under my care at the Western General Dispensary.

August 22nd. Patient is a well-made young man, married and the father of children, habits steady and temperate, complexion sallow, but clear. Health previous to attack quite good. Eyes noticeably fixed and staring, though this had not attracted his own observation. Sole complaint, a constant headache, the locality of which is indicated by passing the hand backwards from the temple over the left side of the head. The above history given in a plain and straightforward manner. No paralysis or spasm present of whatever kind; all reflexes normal. Fundus of both eyes normal, except that the veins are decidedly turgid (this condition confirmed by Mr. Turner, house surgeon); pupils react to light and accommodation, equal in size. Vision perfect. Urine s.g. 1031, no albumen and no sugar. Pulse 85 in the

standing position, soft and regular. Heart, action quiet, and sounds feeble; no murmurs. All bodily functions in good order. Gonorrhœa 16 years ago; no history of syphilis.

Diagnosis: epilepsy, with a suspicion of meningeal hæmorrhage or syphiloma. Ordered pot. bromid, gr. x., pot. iod., gr. v., ter die.

August 29th. Headache better; no more attacks of giddiness, staring of eyes, however, more evident; admits it himself, and says that wife and companions have noticed that he "looked a little wild." Pulse 85. Medicine repeated.

September 5th. Better as regards headache. Exophthalmos now unmistakeable, the upper lid hardly touching the edge of the cornea, and the sclerotic quite visible on every slight movement. Von Graefe's symptom (non-association of lid and eyeball in movement) well marked. Sclerotic not injected, but on the contrary attracts notice by its whiteness, perhaps on account of the sallowness of the complexion. The ophthalmoscope shows nothing abnormal, the turgidity of the veins having disappeared. The thyroid gland, carefully examined, presents no enlargement; it is below the average in size. Heart as before, the pulse always ranging about 85. Mr. Turner also sees the patient, and confirms the condition. Medicine repeated, with addition of pulv. glycyrrhizæ co. for constipation.

September 19th. Exophthalmos somewhat less; Von Graefe's symptom still evident. Headache still continuous, but better. During all this time, has been at work. Vision at no time impaired. Complains, however, of having on one or two occasions during the week, felt for some hours "as if something would happen to him," "as if it would not be safe to get up from his seat." This passes off, and then he feels quite well. At the same time he loses the power of naming things; "knows what they are, but cannot remember their names, and cannot call the children, when he wants them." This also completely passes off. Medicine changed to mist. quiniæ et ferri, bis die.

October 3rd. Seen in my absence by one of my colleagues, who notes the history of venereal affection as above, and that patient continues to complain of much headache. Medicine replaced by pot. iod. gr. v., decoct. sarsæ co. 3i. ter die.

October 10th, on my return. Patient a good deal better for the change of medicine; exophthalmos subsiding; Von Graefe's symptom gone. Headache at times, not troublesome. About this time, on a date which I have forgotten to note, patient's vision was examined by my colleague, Mr. Lindsay Johnson, and

found quite normal. No recurrence of the other symptoms. Pot. iod. increased to gr. x. ter die.

October 17th. Much better; headache ceased, and exophthalmos almost gone. Medicine repeated.

Patient did not attend again.

Remarks.—The prominence of certain local symptoms made this case rather obscure. I have, however, no doubt that it was one of ordinary epilepsy, in which one attack of the major disorder, was followed by several of the minor kind of a somewhat peculiar character.

The point of interest for which I desire to record the case, is the supervention of typical exophthalmos, unaccompanied by any other of the usual signs of vasomotor disturbance. The non-association of lid and eyeball in movement was well marked, and lasted for two or three weeks. Iodide of potassium seemed to be of decided benefit, though this drug has generally not much reputation in epilepsy. As bearing on both epilepsy and exophthalmic goitre, I think the case may be of interest.

A CASE OF OPHTHALMOPLEGIA, WITH GRAVES' DISEASE.

BY ARTHUR MAUDE, L.R.C.P., M.R.C.S.

A MARRIED woman, born in 1845, has had goitre ever since she was eighteen years old. I first made her acquaintance in 1887, but was for some years doubtful as to the existence of Graves' disease, but by the early part of 1891 there was no question as to diagnosis, almost every symptom except exophthalmos being present from time to time. The tremor was especially marked and there was distinct retardation of movement of the upper lids.

We are not concerned here with the general history of the case; a full account of it will be published hereafter with that of other cases.¹ But it seems right to add a note of its peculiar features to those of the other two cases of ophthalmoplegia already published in *BRAIN* (vol. viii., 1886, p. 313, and 1890, Part III.).

On May 11th, 1891, she had a severe crisis of diarrhœa and sickness, such as she has frequently.² She complained of ear-ache on the left side: I found some external catarrh, the membrane dull, and the Eustachian tube closed. There was clearly some middle ear catarrh, but no perforation. The signs of catarrh had passed off by May 15th.

Far from there being any proptosis or retraction of the lids, she has had for years very drooping lids, and the eyebrows are raised habitually as if to counteract slight permanent ptosis.

May 17th. Another gastric crisis. To-day she suddenly showed facial paralysis on the left side, affecting the superior division of the nerve so that the left eye remains permanently open; in spite of this there is no retraction of the lid.

¹ St. Bartholomew's Hospital Reports for 1892.

² I have contributed a note on the peculiar features of such crises to *Practitioner*, September, 1891.

The drooping of the right upper lid is now much more marked.

The pupils are equal and act well to light and accommodation.

May 24th. Facial paralysis continues unchanged. I find to-day that she is quite unable to turn the left eye outwards, and that upward movement of that eye is also very feeble and halting.

May 25th. General left ophthalmoparesis. The left eye lags behind the right in all movements, the muscles seeming to pause occasionally and then act again, bringing the ball slowly into the desired axis. And if she seeks to maintain the deviated position of the balls more than a few seconds, the feeble left side muscles yield and the eye rolls into the state of rest irrespective of the position of the right eye. The pupils normal as before.

This condition remained till June 2nd when the facial paralysis disappeared, and a few days after the ophthalmoplegia disappeared also.

By June 9th all the paralyses were absent, and Graefe's sign reappeared in both eyes.

On June 13th she had distinct exophthalmos. The vision of both eyes was $\frac{6}{9}$ during these weeks. The ophthalmoscopic appearances were normal.

There are details in this case which seem to me to throw light on the pathogenesis of the eyelids symptom in Graves' disease, which I have not given completely here and which I reserve for a further and fuller consideration. I wish, here, only to fix attention on the evidence pointing to some lesion travelling forwards from the fourth ventricle to the aqueduct of Sylvius.

It may be urged that the facial paralysis was an accident due to the occurrence of otitis media.

This I cannot deny; inflammation of the middle ear has been recorded several times in Graves's disease,¹ and in some of them it seems merely accidental. But in this instance the inflammation was very slight and transitory, and apparently passed when the facial paralysis appeared. Moreover, the paralysis cleared up remarkably suddenly if it was caused by lesion in the nerve trunk—while there was no deafness accompanying it.

On these grounds I am inclined to ascribe the conditions to some lesion travelling along the floor of the fourth ventricle close to the middle line, affecting the nucleus of the facial part of the seventh and the sixth nerve first, but leaving untouched the auditory nucleus which lies further out. Moving onwards it

¹Dr. Finlayson. *BRAIN*, 1890. Autumn No. 383. Dr. Bristowe's "Diseases, of Nervous System," p. 69. I have also notes of another case with "Otitis Media," shortly to be published.

reached the nucleus of the fourth nerve, and lastly that of the third nerve.

The paralysis of the third nerve was never complete, and this may account for the curious fact that the pupil was unaffected.

Paralysis of a single ocular muscle in this disease is less common, than general ophthalmoplegia.

Dr. Finlayson's case and that of M. Féréol¹ are the only ones of which I can find details, though Dr. Hector Mackenzie briefly mentions two instances in which he observed "weakness of the external recti producing double vision on looking to the extreme right or left."²

Dr. Finlayson's case differs from that of M. Féréol and mine in the absence of other motor or sensory disturbances which the full reports of the two cases show to have been considerable.

¹ Communicated to the Soc. Méd. des Hôp. Nov. 13, 1874. Given in extenso in various papers—viz., Marie, *Etudes des formes Frustes de la Maladie de Basedow*. (Bureaux Prog. Méd., 1883.) *Bull. et Mem.*, Soc. Méd. des Hôp., 1888, v. *Rev. de Méd.*, 1883, p. 272, and again 1888, 337. (These last are papers by M. Ballet.)

² *Lancet*, 1890, II., p. 546.

Reviews and Notices of Books.

Prichard and Symonds, with Chapters on Moral Insanity.

By Dr. HACK TUKE, M.D. London: Churchill, pp. 116.

THE author of this treatise on moral insanity is to be congratulated on the deft manner in which he seized the opportunities of time and place to honour the memories of these two medical worthies of Bristol on the occasion of a meeting of the Medico-Psychological Association in that city. Dr. Prichard he describes as an enthusiast in ethnology from his earliest years until his dying day. In pursuit of this he acquired French, Italian, Spanish and modern Greek: even as a student at Edinburgh (whither he went after studying medicine at Bristol and St. Thomas's Hospital) this subject was always uppermost in his mind. His work on the "Physical History of Man" (1813) was published a few years after the completion of his medical studies. In it he discussed the question of the races of man being varieties of one species and advanced the theory that the white man was evolved under the influences of civilised life, from an originally dark-skinned race. Dr. Tuke draws attention to the fact that Prichard first "proved the position of the Keltic languages as a branch of the Indo-European" which has since been ascribed to Adolphe Pictet. Prichard saw much of mental and nervous diseases as physician to St. Peter's Hospital, wrote on epilepsy and nervous diseases, became a Medical Commissioner of the Lunacy Board, and created a profound sensation by enunciating the doctrine of so-called "Moral Insanity." Dr. Tuke brings out fully the fact that in Prichard's earliest writings his views were diametrically opposed to this doctrine.

Dr. Prichard appears to have been very successful in the practice of his profession and highly estimable in every relation of life.

Dr. Symonds was born of a puritan family who for five generations had practised medicine. He studied at Oxford and Edinburgh, graduating in 1828.

His tendency to medico-psychology is evidenced by his essays on sleep and dreams, on apparitions, on criminal responsibility in relation to insanity, &c. These Dr. Tuke describes as being characterised by ingenious speculations and original thoughts, conveyed in a singularly lucid and graceful style, quoting several instances of his advanced views on psychologic subjects. Symonds warmly supported the doctrine of moral insanity, supplying Prichard with notes of an illustrative case.

He was distinguished by his practical sagacity as a physician, as well as by his breadth of culture and social qualities. He died in 1871, widely loved and lamented.

Dr. Tuke, having sketched the life and work of his two worthies, devotes the rest of his pages to the subject of Moral Insanity. In regard to this name, Dr. Tuke expresses the hope that a happier term may yet be found: it is certainly objectionable as well from excessive comprehensiveness as from the variety of conditions that, from time to time, have been described under it.

Of the various terms propounded, that of Emotional Insanity, given by Bucknill and Tuke, is certainly misleading; Dr. Rush's Anomia might be admissible, but Parigot's "Diastrephia" is insufficient. The *Manie raisonnée* of Pinel, *Monomania affective*, instinctive or *sans delire* of Esquirol, the *folie d'action* of Briere de Boismont, or the criminal instinctive of Garnier, are all unsatisfactory.

The general recognition of a condition of mental disorder, such as that described by Prichard, is evidenced by this multiplicity of terms; indeed, few alienists of note since his time have not recognised moral insanity, although widely differing in the scope given to it.

Moral insanity cannot be said to have received any practical legal acceptance, although the law has greatly advanced, on the converse side, in recognising that moral responsibility may exist where much intellectual defect is present.

Irresponsibility, where admitted by the law, would often seem to be based on the existence of moral rather than of intellectual defect, but the presence of the latter must always be proven.

If, therefore, moral insanity is a fact, a definite presentment of the subject, such as our author makes, is highly desirable in the interests of justice.

Dr. Tuke quotes the following description of Prichard's idea of moral insanity (page 14) as "the mental state of persons who betray no lesion of understanding, or want of the power of reasoning and conversing correctly upon any subject whatever, and whose disease consists in a *perverted* state of the feelings, temper, inclinations, habit and conduct." Dr. Tuke himself describes the condition as being "not loss of memory, not delusion or hallucination, not any deficiency of talent or genius, not any lack of mental acuteness, and certainly no incoherence of ideas or language—but, a deficiency or impairment of moral feeling or *self-control*, such being either the development of a character natural to the individual, or a departure from it, which contrasts most strikingly with its former traits."

It will be seen that he enlarges Prichard's description by introducing the loss of "self-control"—and he farther dwells on the defect of "inhibitory" power over the impulses and instincts.

The examination of the cases recorded and the conditions described under the term yields as the most typical form, cases that are congenitally deficient and might therefore be termed congenital moral imbeciles. Many such have been mentioned or described, but no recorded case is equal in fulness of life history and consistency of mental state to that reported by Dr. Tuke at the end of this work.

The symptoms-complex of these cases may be thus summarized: History of neurotic heredity, often precocity of intellect and of sexuality. Lying, stealing, cruelty, mischievousness, and destructiveness are manifested from childhood, and are not corrected under the influence of fear or punishment, hope or reward. After puberty they indulge their sexual propensities shamelessly and uncontrollably, leading them on to crimes, and often they become uncontrolled drunkards.

They are devoid of, or only possess rudimentary affections, and are incapable of veneration or the higher emotional feelings.

Their intellect is often described as sharp and precocious; they are plausible, specious and cunning. Although their intelligence is not demonstrably defective, it may yet be considered deficient in the higher planes.

Physically they have not the low development that Lombroso and Benedikt ascribe to essential criminals, but some at least present little physical abnormality.

Their life history may furnish evidence of excitability (from febrile, mental or alcoholic stimulation) and they often end by

becoming intellectually insane. The existence of a numerous class of such cases is confirmed by Dr. Nicolson, who points out (page 61) that a great many criminals, not insane enough for asylums, are unable to be dealt with under ordinary prison rules, and ultimately become inmates of asylums. The Criminal Instinctive of Dr. Garnier would be allied to these, for whose treatment he advocates a special institution midway between the asylum and prison.

A second class is also obvious in which there is acquired defect. In these the moral characteristics have been normal up to a certain period of life when, after an attack of fever, an injury of the head, epilepsy, attack of mania, &c., there is a total change of character. In these moral dementes, as they might be termed, there is utter untruthfulness, dishonesty, and general loss of moral character, evidenced by indulgence of the feelings of the moment and often by indulgence of vicious habits and animal propensities.

There is usually also some loss of mental ability, the individual is no longer capable of the highest mental effort, and his attention is easily fatigued. He is easily excited by drink or mental stimulus, resembling Lesègue's "*cérébraux*" in this respect.

This condition may persist for years, but often it is only a stadium in the development of more marked disease, as general paralysis, senile dementia or insanity.

The indulgence of a habit (alcohol, morphia, &c.) ends in a moral deterioration or dementia, almost identical with the preceding form.

These habits, the result of neurotic inheritance, the cravings of unsatisfied activities or the mere growth of custom, may be regarded as not originating in the higher planes of cerebral development, these being only secondarily affected. That these unfortunates are morally insane in regard to their habit cannot be doubted, and the recognition of this fact is rapidly progressing.

Other cases described under the term moral insanity are those in which the moral faculties are existent but ineffective: the higher levels of the brain are unimpaired, but their activity would seem to be inhibited by the over action of lower levels.

Thus Dr. Tuke examples a condition in which peripheral irritation gives rise to uncontrollable sexual or other impulses, which subside on the removal of the cause.

The cases of premature evolution of sexuality, with various

perversions, such as described in many of Krafft-Ebing's cases,¹ who otherwise have correct moral feeling and normal intellectuality, should probably be classed with these. Their moral defect would appear to be chiefly in the plane of their sexual impulses: although this may lead to general moral deterioration. It is questionable if these Sexual Psychopathics should be held irresponsible.

In the condition of "irresistible impulse" which Dr. Tuke, following Prichard and Heinroth, includes under moral insanity, the moral perceptions are often not only existent, but in active and direct antagonism to the "impulse."

Such cases are numerous recorded and are commonly associated with epilepsy. The example given by Pinel, which he terms *Manie raisonnée*, is of this character.

The impulse may be persistent for years, as in the autobiography recorded by Dr. Manning,² or may be recurrent or transient, often ending in intellectual insanity.

The moral irresponsibility is undoubted, whatever view may be taken of their mental state.

Cases of uncontrollable passion (as anger, jealousy, &c.) are also described. Dr. Tuke quotes an illustrative case of this form, that of a lady who had committed a homicidal assault, and whose violent temper from childhood, and intense jealousy (inherited from her mother) coloured her estimate of others, leading her "to think they were inimical to her."

Pinel³ describes a similar case as *Manie sine delirium*. This man was described as being not only highly intelligent, but also beneficent and compassionate. The irresponsibility of such persons may be beyond doubt, but it is questionable whether the plea of maniacal excitement would not be sounder than that of moral insanity.

Dr. Tuke appears to include also states of abnormal emotional change without delusion. In this he follows Prichard, Guislain and Bucknill, who have all pointed out the existence of moral perversion with emotional depression (*melancholia*) without delusion. Pinel's reasoning madness and Esquirol's affective monomania appear to describe a similar condition, in which there might be emotional exaltation, rather than depression.

In protracted cases, these are said "by plausible emotions, by reasonable explanations, to gratify their sentiments and excuse the strangeness and inconsistency of their conduct."

¹ "Psychopathia Sexualis."

² *Journ. Men. Scien.* 1882.

³ Trans. by Davis, p. 151.

The condition is that of perversion rather than loss of the higher sentiments, and there are certainly associated changes of emotion and of intellect.

Dr. Tuke (p. 91) quotes the opinion and reasons of Herbert Spencer for believing the possibility of the existence of moral without intellectual defect. He (Spencer) argues "that although intellect is evolved out of feeling, and cognitions are inseparable from feelings," yet there may be "varying degrees of relative development of emotion and cognition."

Practical illustrations of the abnormal relations between cognition and feelings are indeed constantly observed in early states of cerebral disorder, as in melancholia. The incipient melancholic constantly attributing to himself moral blame, that accustomed cognitions are no longer associated with their wonted feelings.

This variability, however, does not affirm the complete separability of feeling from cognition, and nothing less than this would be sufficient to prove the possibility of moral without any intellectual defect. Admitting the varying development of feelings and cognitions, the conjecture may be made, that while the one may be recorded with little defect, the other may be so imperfectly registered as not to be available for the complex aggregations of feeling of which the highest sentiments are compounded, these consequently remaining undeveloped, moral imbecility being the result.

The predominant disorder of emotion, in the earliest departures from mental health, has long been recognised by Sankey, Guislain and Bucknill, and is evidenced, as Dr. Tuke quotes, by the evanescent states of depression produced by some states of health and by drugs, as hyosine, &c., while the disorder of the more complex moral feelings, from more prolonged disorder of the cerebral nutrition, is evidenced in the variations in character, often accompanying the evolution of puberty, menstruation, gestation, and other physiological conditions.

From these considerations it might be deduced, as Dr. Tuke suggests, that a stage might be found in every case of insanity in which the higher emotional disorder might be predominant, the intellectual defect being scarcely appreciable.

The writer of this article is quoted (at page 113) as recognising the occurrence of such phases, of greater or less duration, in various mental diseases. If, therefore, these may occur episodically, there is little reason to doubt their occurrence as persistent conditions.

In such cases as Dr. Tuke writes, the higher levels of cerebral evolution, the seat of the most voluntary functioning with which the altruistic sentiments are associated, have become the seat of morbid changes, leading to their inaction and the increased activity of the lower areas by the loss of their controlling influence. These cases of secondary defect might well be termed, as already suggested, acquired moral dementia or imbecility.

The cases of defect, whether from the non-evolution or loss of function in the highest and most voluntary areas of the brain, assuredly do not correspond either psychologically or in probable cerebral localization with those in which the moral feelings have been evolved and are still active, but ineffective.

The predominance of an animal instinct, the insistance of a habit, the overpowering stimulus of passion, the "irresistible impulse" of disease, are examples, as Dr. Tuke recognises, of the highest centres, although existent and active, being unable to control the abnormal activity of lower cerebral planes.

The two classes of defect are, no doubt, widely separated, as Dr. Gasquet¹ points out, in psychological significance; in the one class the higher centres are either not evolved, are degenerated, or temporarily functionless; in the other they are developed, potent, and exercising function, although ineffectually, being inhibited by the hyperactivity of lower centres.

These latter states may be, for convenience, classed under the head of moral insanity, from the fact of the resulting actions, but the intrinsic psychological difference should not be thereby obscured. These might be justly considered cases of moral perversion, but are certainly not moral imbecility of either form.

From this consideration of the clinical facts recorded, and of their psychological aspects, Dr. Tuke's conclusion may be fairly accepted that there "are morbid cerebral conditions, in which the mental symptoms displayed are the emotional and most automatic rather than those concerned in cognition," and that these "may be referred to the form of mental derangement usually called moral insanity."

The several varieties to which he alludes might be fairly grouped into three classes. First, the congenital moral imbeciles, in whom the moral faculties have never been evolved; secondly, the acquired moral imbeciles, in whom they have been lost; and thirdly, cases of moral insanity, in whom they are existent, but are perverted, or are incapable of exerting control.

¹ *Jour. Mental Sci.*, 1882.

That the plea of irresponsibility in the two first classes may be fairly claimed on the ground of moral defect can be readily conceded, but in the latter class it is doubtful whether, in a large proportion of the cases, this should constitute the whole plea the defects being secondary to demonstrable emotional, and even intellectual change.

Moral insanity has to be differentiated from lunacy on the one hand and moral depravity on the other.

Every alienist has experience of the great moral perversions in mental disease, and the predominance which they often manifest over the intellectual disorder. A case recorded by Dr. MacFarland,¹ in which a delusion concealed for twelve years produced conduct that might have been regarded as typical of moral insanity, illustrates the necessity of caution in assuming the non-existence of intellectual defect.

The differentiation, from moral depravity, Dr. Tuke justly concludes, can not be made by any rule, "each case must be decided in relation to the individual himself, his antecedents, education, surroundings, and social status, the nature of certain acts, and the mode in which they are performed, &c."

The medico-psychologist may from this study of the individual arrive at a definite opinion, but it is much more difficult to satisfy a judge and jury of the irresponsibility of such cases.

The treatment of the conditions is only slightly touched in these pages, but it would certainly be affected by the suggestion which Dr. Tuke makes that the congenital cases are atavistic. The writer would suggest that this is an incorrect view, calculated to lead to unhopefulness. Should they not rather be considered examples of abnormal evolution who would be abnormal in any society or stage of civilization. It appears as unjust to compare them with savages, as it is to liken a depraved man to a beast.

Deprivation of education, as suggested by Dr. Kerlin, does not commend itself, and it is probable that the most successful procedure would be a careful attention to physical health with a very special and individualized system of moral training. How many individuals, under favourable conditions, are thus trained to escape, more or less completely, the results of their faulty organisation is at present unknown, but the number is probably considerable.

The cases which become most prominent are those in which, as in Dr. Tuke's typical case, the individual has been treated

¹ *Jour. Mental Sci.*, vol. ix., 380.

neither as insane or criminal, not obtaining the treatment of the one and only the evils of the punishment of the other.

In regard to the allied forms of moral defect considered, it is only necessary to insist that they should be treated as insane, and not as criminals.

Dr. Tuke is to be congratulated in having brought forward his subject so clearly and thoughtfully, and the attention of medico-legists will be certainly in future more fully directed to this important question.

HENRY RAYNER, M.D.

Les fonctions du Cerveau. Par JULES SOURY, maitre de conférences à l'Ecole pratique des Hautes Etudes.

FOREMOST amongst the contributions of the laboratory to practical medicine stands the discovery made in 1870 by Fritsch and Hitzig, of the excitability of the cerebral cortex; together with the corollary added by them, and subsequently elaborated by Munk, Ferrier, and others, that all portions of the cortical grey matter are not equally irritable. That results of so great practical importance should provoke much controversy, partizan as well as critical, was inevitable; and the accumulation of literature, after some twenty years of publication alike of facts and fancies, is enormous. The contribution to this fund, which lies before us, is from the pen, not so much of a physiologist as of a *littérateur*, and is a critical history of the advances made in cerebral physiology, since the important observations to which we have above alluded.

The work is composed of papers reprinted from *l'Encéphale* and *les Archives de Neurologie*, and is presented in one volume. It comprises two main divisions, which deal respectively with the teachings of the Strasburg laboratory, on the one hand, and on the other with the tenets of the Italian schools. We regret this purely arbitrary, and, *ipso facto*, unsatisfactory classification of contemporary work, which the author, after some deliberation, has adopted; and we fear that the double reference and repetition necessitated by his procedure, will repel, rather than invite, the student of brain-physiology. We will pass, however, from criticism of the volume, to analysis of it, treating it as a single, not as a double compilation.

The functions of the cortex have been studied by the complementary methods of excitation and destruction, both of which

have demonstrated marked difference of function between different regions. To both methods various objections have been raised. Against the first it has been urged that electrical stimulation of the cortex involves, by means of current diffusion, simultaneous stimulation of the subjacent white matter. But it must be remembered, firstly, that the corona radiata is less easily excitable than is the cortex (Putnam); secondly, that currents of workable strength do not diffuse laterally far enough to stimulate regions 1 mm. distant from the nearer electrode (Rouget), and therefore, presumably, would not affect the more distant, and less irritable corona; and thirdly, the period of latency in the case of cortex-stimulation is distinctly longer than that involved by corona-excitation (Pitres and Franck, Heidenhain and Bubnoff). Lastly, the cortex is said to be excitable by mechanical (Luciani) and chemical (Landois) stimuli. Further evidence on these points is, however, desirable.

With regard to the second method, that of destruction, two sets of symptoms accompany its application. The one set are possibly due to the inhibitory effects of the operation, upon apparently uninjured territories (inhibitory symptoms) and are transitory in duration. The second set are referable to the loss of the destroyed area (ablatory symptoms), and are partly transient, partly permanent. They are partly transient because, after a while, the uninjured convolutions of the same side, together with the corresponding portion of the opposite hemisphere, and the sub-cortical grey matter (basal ganglia—Ferrier, Luciani), are able, in a measure, to take upon themselves the functions of the injured area.

Hence, it is not easy to distinguish a transient ablatory symptom from an inhibitory phenomenon (Goltz).

The earlier experiments of Fritsch and Hitzig, had led them to state, that the cortex is excitable at certain points, not excitable at others. Later experimenters (Ferrier, Schäfer, Bechterew), however, obtained movements by stimulating sensory areas. Excitation of the occipital lobes, for example, caused conjugate deviation of the eyes to the opposite side. Similar movements may be elicited by stimulating the frontal oculo-motor centre, but they are said to differ from the results of occipital stimulation, in that they do not require so strong a stimulus, and in their shorter latent period (Schäfer and Horsley). These movements, resulting from excitation of the occipital lobe, are regarded by Ferrier, Schäfer, and Horsley as being reflex in nature, whilst by Bechterew, Luciani, and Danillo they are believed to

be comparable to the results of stimulating motor areas. Hence we are no longer able to speak of excitable and non-excitable regions, though the difference in excitability between different areas is very great. In brief, the results of the method of excitation point clearly to marked difference in function between different regions. Not to perfect homogeneity, nor to sharply-defined localization, but to concentration of certain functions in certain regions. The connection of each hemisphere with the body is bilateral in nature, for, whilst the results of weak stimulation are frequently confined to the opposite side, strong excitation always provokes bilateral response.

The results of localized brain-destruction tend to advance the above quoted views, with regard to concentration of function, as well as to corroborate the theory of the bilateral connections of both halves of the brain. For, as we have mentioned above, the residue of symptoms, after disappearance of the inhibitory effects of the operation, is not, *in toto*, permanent, but gradually diminishes as compensation is established. Ablation too, affords us information on sensory areas that stimulation cannot give. Removal of the so-called motor area is frequently, not to say generally, followed by sensory symptoms of a paretic or paralytic nature. And this, not merely of the sense of touch, but also, though possibly in less degree of the senses of temperature-change, of pain, and of muscular sense. English opinion seems to point to the limbic lobe as being especially tactile, &c., in function; whilst German, Italian, and French teaching would indicate that these (tactile, &c.) centres are nearly co-extensive with those of motion, though less concentrated in distribution than the latter. To produce *definite* sensory symptoms, a more extensive ablation is necessary, than to produce well defined motor disturbances, and these (sensory) symptoms, when produced are rather paretic than paralytic in nature. In favour too, of the sensory functions of the motor area, we would adduce the frequent occurrence of sensory *aurae* before the motor manifestations of Jacksonian epilepsy. *Sensori-motor*, then, would be a better attribute for these regions than is simply *motor*. But extensive ablation of these areas is accompanied not merely by sensory, and motor phenomena, but in addition by mental changes. A dog, deprived of both motor areas, may recover, to a large extent, his powers of movement, and sensation by means of his uninjured cortical and sub-cortical grey matter. But *mentally*, he apparently never recovers. He has forgotten all his *tricks*. He will no longer give a paw, nor beg for food. In every way he is stupid. The area destroyed was *psycho-motor* in function.

From the regions that are more particularly motor in function, we now pass to those more especially sensory. Extensive cortical lesions, in dogs, are always associated with visual, and auditory troubles (Goltz); the former being more pronounced when the lesion is posterior in position, the latter when it is situated laterally (Luciani and Tamburini). We will first deal with the visual functions of the cortex, which are concentrated at the posterior end of the cerebrum (Munk, Luciani and Tamburini). The importance of the cortical visual area varies greatly in different species of animals, increasing as we ascend from lower, to higher types. Frogs and fishes deprived of both hemispheres can see, and see intelligently (Goltz, Blaschko, Schrader). Pigeons under similar circumstances suffer more severely at first, but still, recover more or less intelligent vision (Goltz, Schrader). Dogs deprived of both visual areas exhibit still more severe transitory symptoms, and *never* recover psychical vision, and in monkeys the effects of similar lesions are analogous though still more pronounced. In dogs and monkeys *complete* blindness has been stated to follow bilateral extirpation of the visual centres (Munk), but Luciani has never succeeded in inflicting other than psychical blindness, by cortical lesions. The connection of each visual area with the organs of vision is bilateral (Munk, Luciani), the effect of any unilateral injury being hemianopia, or hemiamblyopia, according to the extent of the lesion.

The auditory area, in dogs and monkeys, is placed by Luciani and Tamburini in front of the visual region, and auditory functions are most concentrated at its lower extremity. Unilateral extirpation of this area gives rise to bilateral deafness, more pronounced on the side opposite to the lesion, and transitory in duration. Ablation of the second area makes the deafness equal on both sides and more permanent in duration. But gradual recovery occurs in all cases. (Luciani and Tamburini).

Olfactory and gustatory troubles frequently follow posterior and lateral lesions in the dog (Goltz). Luciani and Tamburini believe, that the region in front of the auditory area, in dogs, is especially olfactory in function. These three sensory centres—the visual, auditory, and olfactory—are even less sharply separated from one another than are the motor centres. Each one diffuses far into territory covered also by its neighbour.

We have already cursorily noted psychical troubles following bilateral injury to the motor centres in dogs. Extensive bilateral lesions leave behind them, in addition to the clumsiness, and stupidity already mentioned, notable alterations in the *disposition*

of the animal. Anterior lesions transform a quiet, well-conditioned animal into a restless, ill-tempered creature, very prone to lose flesh. Posterior lesions, on the other hand, convert snappish and lean animals into inoffensive creatures, with a tendency to grow fat (Goltz).

We would here allude briefly to some important statements made by Mosso in relation to cerebral activity. This observer has noticed, in three human beings in whom a great part of the frontal portion of the calvarium was lacking, that during sensation, emotion, or thought, the volume of the forearm (plethysmographically registered) decreased noticeably, whilst that of the brain underwent an equally obvious increase. These experiments, which seem to show a marked congestion of the brain during cerebral activity, lend an indirect support to some observations of Schiff's, who has observed, with the thermopile, a rise of temperature under such circumstances. Corso, on the other hand, has noticed a fall of temperature more frequently than a rise under analogous conditions. Tanzi, who tested the meninges and not the cortex, describes sometimes a rise of temperature, sometimes a fall, with cerebral activity. The rise of temperature has been attributed to synthetic chemical action in the grey matter during comparative rest, and the fall to the analytic chemical changes that accompany brain-work. We would point out, however, that vaso-motor changes constitute a fruitful source of fallacy in such interpretations.

Much has been added of late years to our knowledge of the anatomy of the central nervous system by the improved methods of Weigert and Golgi. The latter histologist, by treating specimens hardened in bichromate of potash, with silver nitrate or corrosive sublimate, has succeeded in staining the nerve cells and their processes with a distinctness not previously attained. As a result of his labours he has seen a branching of the cell-processes, far exceeding in complexity anything before imagined. Of cell-processes he describes two varieties: the one, "*nervous process*," of which each cell possesses only one, becomes the axis cylinder of a nerve fibre, whilst the second set, "*protoplasmic processes*," of which each cell possesses many, form a dense network (*réseau*) in the grey matter. We regret that the attribute "*protoplasmic*" has been applied to the processes of the second type, such nomenclature implying the non-protoplasmic nature of the others. Of this non-protoplasmic nature of the nervous processes, we have no proof and we shall refer to the non-nervous branches of the cells as *nutritive processes*. After

long seeking, Golgi has confirmed the statements of Kölliker, Krause, Deiters, and others, that no anastomoses between the cell-processes can be seen. They are in intimate contact with one another, but no direct junction between two cells has ever been observed. The *nutritive processes* run chiefly towards the surface of the brain and are in intimate connection with the neuroglia cells and with the walls of the blood vessels, whence their name.

The *nervous processes* of which, as we have before stated, each cell possesses only one, at a variable distance from their cells, branch richly, and from the nature of their branching two types of *nervous processes* may be distinguished. Nervous processes of the first type, though, laterally, they branch richly, do not lose their apparent individuality: whilst those of the second type, branch not only laterally, but also longitudinally, so much as to quite lose their apparent individuality. Nervous processes of the first type are joined to cells which resemble, in appearance, those of the anterior horn of the spinal cord, and which consequently are perhaps motor in function. Processes of the second kind are united to cells like those found in the posterior horn of the spinal cord, and which, therefore, are possibly sensory in nature. Further, the motor cells are by far more numerous in the more especially motor centres; whilst the possibly sensory cells occur to greatest extent in the more sensory regions. At the same time both varieties of cells occur everywhere in the cortex, those of the sensory variety being more superficial in position. In relation to this hypothesis of the nature of the two kinds of cells, we may here note that Lissac has stated, that superficial lesions may produce more definite sensory than motor symptoms, whilst deeper lesions are accompanied by better marked motor paralyses.

With regard to the basal ganglia, the supposition is not in accordance with the teachings of most observers. For V. Marchi has found, that in the corpus striatum, especially in the caudate nucleus, cells of the sensory type are most abundant, whilst in the optic thalamus, motor cells occur in greater quantity than the others.

Our analysis of M. Soury's work is far from complete; but it is sufficient to indicate the wide extent of the author's reading. M. Soury shows an intimate acquaintance with the literature of Physiological Neurology, and a strong bias in favour of Italian opinion. The work is attractively written, but the diffuseness of the author's style, added to the unsatisfactory classification to

which we have above alluded, renders reference a matter of difficulty. To the reader, with sufficient time at his disposal, we can recommend M. Soury's work as an intelligible, and picturesque account of Neurological research.

WILLIAM LEGGE SYMES.

The Intra-Cranial Circulation, and its Relation to the Physiology of the Brain. By JAMES CAPPIE, M.D.
Edinburgh : James Thin, 1890.

IN this volume Dr. Cappie has re-stated and amplified his theory of the "Causation of Sleep," and under its new title, it will be found that the argument, for such it claims to be, has lost none of its point and vigour. Maintaining that diminution of cerebral arterial blood during sleep is compensated by increase of venous blood, and that the amount of blood within the cranium is under physiological conditions, invariable, he excludes the cerebro-spinal fluid from any share in the compensation and the vaso-motor nerves from any part in the mechanism. This is the nucleus of the volume. While appreciating and admitting the importance of experiment, his own method, he says, is not experimental. He does not, however, on that account, claim for it exemption from appeal to experiment where that is possible, but, on the contrary, invites such a test.

In the Chapters on the "Philosophy of Physical Causation," and the "Correlation of Mind and Brain," the dialectic is skilful and interesting. Assuming the cranium to be a closed box with its contents not directly subject to atmospheric pressure, the author, stoutly applies purely physical laws, as complimentary to his deductions from the experiments of others, in an endeavour to maintain the empirical doctrine of the uniform mass of blood within the cranium.

There is a certain heroism in his regard for the second Monro and Kellie of Leith, but the statement "that the encephalic circulation receives less attention than in the early years of the century," although a tribute of the author's admiration for the work of these writers, is scarcely accurate, and implies a quite unmerited reproach to modern research in a subject which owes so much to the recent developments of the injection method. The method, however, that has shed so much light on cerebral vascular distribution is not once referred to in the volume. Neither is

there any evidence of acquaintance with such modern works on the cerebral circulation as those of Heubner, Duret, Charcot, Cohnheim, and other well-known names, while the views of Northnagel and Krauspe on the relation of nerves to arterial contraction are completely ignored.

In discussing the circulatory mechanism Dr. Cappie expresses scepticism regarding the action of vaso-motor nerves.

"Vaso-motor nerves," he observes, "appear to get credit for being veritable physiological demons. Their unsleeping vigilance forsee and provides for all local wants. They so regulate the calibre of the smaller arteries, that they turn on, or shut off, the blood current as may seem to be necessary."

The cerebral vaso-motor nerves may, however, claim that the "current" estimate, instead of being extravagant and far fetched, errs on the side of modesty. For Burekhardt has demonstrated that the well-known rhythm common to arteries all over the body exists also in the brain, and in its modifications reflects the characters of even mental processes; while Goltz, Ditmar, and others have shown that increased arterial pressure keeps pace with sensation.

It is not, therefore, strictly in accordance with our knowledge to observe that "*the encephalic circulation will be focussed in the direction of activity*," without a qualification that would recognise the character of the activity as the determinant of blood pressure.

The heart, he regards, insufficient in itself to carry on the circulation, is aided "by a local molecular action in the tissues" — *a vis a fronte*, as *e.g.*, in carrying the blood through the hepatic capillaries to the hepatic veins, inferior cava, and right auricle. "It is obvious," he argues, "that no influence of vaso-motor nerves can in the least assist us here." But the pulse alone, we would observe, is evidence sufficiently crowning that the blood in the arteries is kept under pressure by vaso-motor action, while there is a great mass of experimental evidence which compels us to regard the arterial movements as controlled and regulated by a nervous mechanism. Veins are similarly controlled, although to a less extent, for, after section of the medulla, as was shown by Schiff, the veins relaxed so much as to require the blood of another animal as large as the one experimented on, to raise the pressure. During diastole of the left ventricle then, the arteries contracting on the blood they contain, keep it flowing on through the capillaries till the next systole, and, as the author concedes at another place, "with the least tendency in the right auricle to dilate a suction force is exerted." This then is a *vis a fronte*.

The very possibility, therefore, of the circulation depends on the effectiveness of the vaso-motor influence, as Marey has observed, "the rate of the heart is in inverse ratio to the arterial pressure," and the latter, as we have shown, depends largely on vaso-motor conditions.

Dr. Cappie, as an able exponent of the doctrine of Monro and the experimental conclusions of Kellie, maintains the thesis "that the mass of blood within the cranial cavity can be neither diminished nor increased directly, and that the extent to which it can be altered by ordinary physiological causes within short periods of time must be very limited." Sir George Burrows opposed this doctrine of Monro and Kellie, concluding from a series of experiments on rabbits, that bleeding diminished the quantity and momentum of blood in the brain, and that, as a rule, the brain is congested after death from any form of asphyxia, or interference with the return of venous blood. Further, that diminution of one system of vessels does not necessarily imply repletion of the other, and that circumstances vary the amount of blood within the cranium, but that "blood, brain, and serum together, must be at all times nearly a constant quantity." In investigating the same subject, Donders closed air-tight with a piece of glass an opening trephined into a rabbit's skull, and with a microscope, observed the vascular changes. Kussmaul and Tenner repeated and elaborated the experiments of Donders, and confirmed his conclusions. On closure of the left sub-clavian artery and compression of the innominate they observed the brain become pale, the smaller vessels invisible, and contraction of veins opening into the longitudinal sinus, with a doubtful contraction of the sinus itself. On closure of the nostrils, or on convulsions supervening the veins enlarged, but the position and pallor of the brain remained unchanged, continuing so till after death. On restoring the circulation the brain became pink, the finest vessels visible and the veins enlarged. In the opened cranium the vascular phenomena were the same, but on closing the nostrils, or on convulsions supervening the brain swelled without turning red, although the veins on its surface enlarged.

The experiments of Kellie to which Dr. Cappie attaches so much importance were conducted on sheep and dogs. He concluded that the brain vessels are not emptied by any means of general depletion as vessels elsewhere are, although profuse hæmorrhages drain it "of a sensible portion of its red blood"—the place of the blood being taken by serum. He opened the

cranium of a dog and bled the animal to death. The brain subsided and contained very little blood, whereas in the unopened skull the brain filled the cranium and contained "a considerable quantity of blood."

The conclusions of Kellie, therefore, and those of the eminent observers who followed him, although differing in degree, subscribe to the same doctrine—that while the absolute contents of an intact cranium continue to fill the cavity, the proportion of blood may vary.

In cases of death from hanging, Dr. Cappie observes, that while the surface veins may be gorged, no impression is made on those within the cranial cavity. The statement is entirely opposed to the conclusions of Burrows, and to such authorities as Casper, Renner, and others who record that cerebral congestion and even apoplexy may be present. He defends the conclusions of Kellie from the charge of affirming the invariableness, in all circumstances, of the amount of blood within the cranium, although the justification of Kellie would seem fatal to his own theory, yet, notwithstanding this qualification there is an eloquent contention throughout the work for the uniform mass of blood within the cranium. If then the amount of blood within the cranium be constant, it must follow that the cerebro-spinal fluid does not flow between the cranial cavity and spinal canal, for the brain mass being constant or nearly so, variations in the mass of one fluid would imply a compensatory variation in the other. If, he observes, the fluid can be so squeezed into the spinal canal something there must make way for, and the brain being anchored by twelve pairs of nerves, the ebb and flow would mean a moving of the brain which "would resemble an instrument with slackened strings," from the absence of stress or support necessary to the discharge of nervous energy. There is no evidence that the analogy is true, for open fontanelles do not interfere with cerebral operations, nor does isolation of a nerve prevent the transmission of impulses. That the brain although anchored moves, and that the fluid ebbs and flows by virtue of this movement is no paradox, for Burckhardt on four cases of defective skulls obtained tracings of three phases of brain movement: (*a*) The vaso-motor wave common to arteries (the vascular wave of Mosso) 2-6 per minute; (*b*) pulse wave 60-80 per minute; (*c*) respiratory wave (Ecker) 15-20 per minute.

The behaviour of the cerebro-spinal fluid before this alternate protruding and depressing force is shown by Quinke, who, from the spinal subarachnoid space injected with cinnabar the

Pacchionian glandulæ, the dura mater, sheathes of cerebral nerves, and cervical lymph glands.

These experiments Burekhardt maintains prove the vascular wave to be a motor mechanism designed to carry off waste products through the lymphatics by setting up currents in the brain fluids. In illustrating how far the cranial contents are removed from atmospheric pressure, the second *Monro*, says Dr. Cappie, used a hollow glass ball filled with water, observing "that not a drop of fluid escaped when inverted with its aperture turned downwards." To perform the experiment on the dried skull, he continues, let the sockets be plugged with incompressible balls, and the fissures and foramina guarded by fibrous tissue, and "just as happens with the glass ball none of the water will be dislodged by gravitation." We would again observe, however, that foramina and fissures are not, in the normal, so secured, but transmit a circulation of fluids; neither are they confined to the base of the skull as the argument and illustration would imply, for the sinuses of the brain are joined by veins from the nose and face, and veins pass from the scalp to the superior longitudinal and lateral sinuses. These communications receive clinical confirmation from the author himself in the observation that binding the head relieves certain forms of headache, for, he observes: "internal support to the brain may be modified by external pressure." It is not, however, readily conceivable that binding the hollow glass ball would so modify its contents, and rather than attribute to the author the imposition of such a difficult task we prefer to assume the analogy as strained. In a chapter devoted to the "Causation of Sleep," Dr. Cappie discusses a theory that is by no means altogether unphysiological. Briefly it is that "the circulation in the brain itself is diminished and to a corresponding extent the blood in the veins is increased," resulting in "compression of the whole organ by hydro-dynamic agency." The *modus operandi* is thus:—on cerebral activities slowing, the brain subsides from the surface of the skull, its retreat being covered by turgescence of the veins of the pia mater, for aided by atmospheric pressure, they recoup themselves from the blood in the veins leaving the skull. The pressure becoming a "compressing" one, and "with a certain amount of pressure consciousness is suspended." It would be difficult to reconcile this theory with the effects of posture on the circulation in the brain.

"In some parts of India," says Brunton, "the natives are accustomed to bring persons round from a faint by compressing the nostrils, and holding the hand over the mouth, so as to completely

stop respiration; a treatment, as shown by the experiments of Burrows and Kussmaul and Tenner, that would increase the blood in the veins by interfering with its return. Granting however, that the brain in sleep is so held down by the engorged veins of the pia mater, the circumstance, as a sequence to diminished capillary circulation, would be no more than a simple item in the series of change the aggregate of which is sleep.

It is, therefore clear, that believing in the uniform mass of blood within the cranium, Dr. Cappie is able to dispense with the cerebro-spinal fluid as a compensatory mechanism, and doubting the existence of Magendie's foramen on the authority of Todd, he regards the theory of compensation from spinal fluid as anatomically improbable, if not impossible. But it has been shown that the perivascular and perineural spaces subserve the function of accommodating the ebb and flow of fluid in obedience to variations in intra-cranial pressure. We are not unprepared to find the vaso-motor nerves share the fate of the cerebro-spinal fluid in his estimate of the mechanism, for he is "not aware that any definite knowledge has been got as to their mode of action," and the compensation, in his theory, being established by purely physical laws would render their action superfluous.

Finally, in the last chapter, that on Some Points in Mental Physiology, to vaso-motor nerves is ascribed the possible function of aiding in preventing the brain from becoming over expanded. In his own words, "lastly there is the delicate balance of check and counter check (in which possibly the vaso-motor nerves may act an important part) between the tissue changes and capillary circulation."

It is very obvious, therefore, that modern research on the subject is not overtaken in the volume, for a few at least of the arguments and theories, once the best of their kind, are so no more; nevertheless, the subject is made interesting, and many better known and more voluminous writers on science than the author might pardonably envy his English style and eloquence.

JOHN CUMMING MACKENZIE, M.B.

Ueber die chronische Progressive Lähmung der Augenmuskeln.
Dr. E. Siemerling. *Archiv. für Psychiatrie und Nerven-*
krankheiten. XXII. Band.

THE author of this Monograph describes it as a "legacy of Westphals." The great neurologist had laid the foundations

of a work dealing with the chronic progressive paralysis of the ocular muscles, and Dr. Siemerling had, from the first, helped him in his anatomical investigations and had prepared his specimens. After Westphal's death, Dr. Siemerling determined to accomplish what his chief had hoped to do himself, and as a result, we have this excellent memoir on a very difficult subject, which the writer has handled in a clear and able manner, and with a vast amount of industry. In addition to his own personally observed cases, which number eight, he has analysed and criticised all the cases which he could find recorded by other writers. In explanation of the text he has given twelve plates, representing, for the most part, the stained macro- and microscopical specimens taken from his own cases. The book is well worthy of the great neurologist, in whose honour it was undertaken, and the best thanks of all those interested in nerve disease are due to its author, Dr. Siemerling. An analytical table of the symptoms and pathological changes present in the eight personally observed cases is given at the end of the book, and can easily be referred to by anyone specially interested in the subject.

The following notes will give some idea of the cases:

CASE I.—All the muscles of the eye were paralysed, and *post-mortem*, atrophy of the centres of the oculo-motor nerves in the pons was found, together with degeneration of the nerves proceeding from them, and of the muscles supplied by them. There was also disease of the posterior columns of the spinal cord in their whole length.

CASE II.—Clinically the symptoms pointed to tabes dorsalis and disease of the lateral columns of the cord as well. Ophthalmoplegia externa and interna, and atrophy of optic nerves, were also present. At the autopsy, atrophy and degeneration of the third and sixth nerves and centres were found and disease of the posterior and lateral columns of the cord.

CASE III. The patient was a painter, who had never had symptoms of lead poisoning, and who had had a chancre, not followed by any eruption.

Ophthalmoplegia, optic atrophy, and symptoms of tabes dorsalis were present.

Post-mortem. Atrophy of optic nerves, and degeneration and atrophy of the third and sixth nerves, their centres, and the muscles supplied by them, as well as disease of the posterior columns of the cord, were found.

CASE IV.—Ophthalmoplegia and partial atrophy of optic nerves, together with dementia, observed during life. After

death the cord was found to be healthy, but the oculomotor nerves, their centres and their muscles were degenerated and atrophic.

CASE V.—Case of cerebro-spinal sclerosis. Ophthalmoplegia was here caused by the interruption by a sclerotic patch of the nerves proceeding from the centres of the oculomotor nerves, these centres being intact.

There was in addition mental weakness, and a very stubborn neuralgia of the right fifth nerve, which was involved in a patch of sclerosis.

CASE VI.—Case of general paralysis. Knee-jerk absent; re-action of pupil to light gone, to accommodation retained. Ophthalmoplegia.

Post-mortem. The centre of the hypoglossus and its efferent fibres were found degenerated, as well as those of the third, fourth and sixth nerves. Great distension of vessels in pons, patch of atrophy in right optic nerve, atrophy and degeneration of ocular muscles, and disease of posterior columns of cord.

CASE VII.—Case of general paralysis. Ophthalmoplegia externa; absence of light reflex. The ophthalmoplegia was much more marked on one side than on the other. The right external rectus was not paralysed.

Centres of fifth, fourth, third and left sixth nerve degenerate, together with their nerves and the muscles supplied by them. Cord intact.

CASE VIII.—Case of dementia. Well marked atrophy of right optic nerve, slight of left. Ophthalmoplegia of left eye, sixth nerve alone paralysed in right.

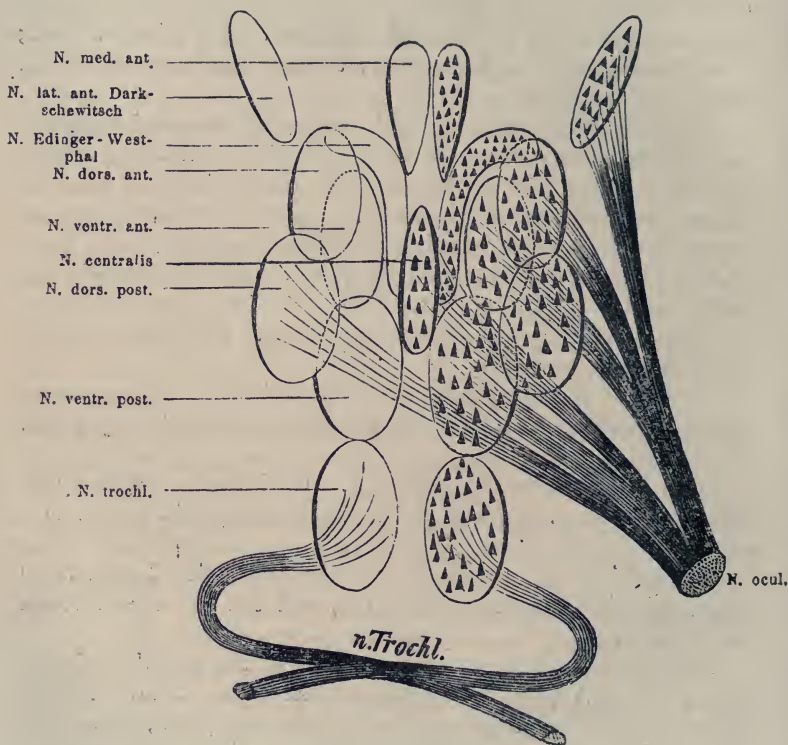
Light reflex gone, convergent reflex present. Later, more muscles of right eye became affected, and the convergent reflex disappeared.

Post-mortem. Irregular degeneration of the spinal cord was found, different parts being affected at different levels. The centre and fibres of the hypoglossus were atrophied, as well as those of sixth nerves, and of third on left; ocular muscles slightly atrophic. Optic nerves degenerated.

The notes of these eight cases terminate the second section of the volume.

Section III. This deals with the results of the investigation of the centres of the oculomotor nerves, and the author shows that in most cases of ophthalmoplegia the disease is nuclear. In all cases the sixth nerve is affected, and nearly always bilaterally, and the same may be said of the third. Dr. Siemerling

shows (as may be seen in his diagram which is here reproduced) that the centre for the third nerve consists of several clearly defined groups of cells, and that decussation of some of their fibres takes place. But in spite of the evident separation into cell-groups we are not yet in a position to connect the functions of the several oculomotor muscles with individual groups, and can only say that, on the whole, it is very probable that in man the centres for accommodation and the movements of the iris are



situated anteriorly, those for the muscles elevating the eye in the posterior and lateral divisions of the oculomotor centre. Moreover, though many investigations have had for their object the demonstration of the path traversed by the reflex stimulus between the optic nerve and the oculomotor centre, we have as yet no accurate knowledge of it.

Section IV. deals with the nature of the pathological process which produces ophthalmoplégia. The conclusion arrived at by

the author is that it is a primary degeneration of nerve cells, as was first proved by Hutchinson and Gowers. Nerve cells in all stages of degeneration are found, diminution in the nerve fibres within the centres, and some thickening of connective tissue. Hyperæmia, hæmorrhages, alterations in blood vessels, ependymitis, &c., are only occasionally present. The actual cause of the degenerative changes in the centres is uncertain. Dr. Siemerling sums up thus the pathological conditions which have been found causing ophthalmoplegia :

1. Affection of the oculomotor centres themselves, together with degeneration of the efferent nerves and their muscles.

2. Affection of oculomotor nerves and muscles with intact centres, which is very rare indeed.

3. Patches of sclerosis in the course of the intramedullary part of the nerve roots, with intact centres and muscles. With the fewest exceptions, ophthalmoplegia results from degeneration of the nerve centres, followed by degeneration of the nerves and muscles. Some observations which have been recorded have yielded absolutely negative results, but the investigations lacked completeness.

Section V. deals with the results of the examination of the peripheral nerves and muscles, and with alterations of other parts of the nervous system, which are found in those cases. Optic atrophy is often observed; and although some have suggested that it is a descending change, there are no good reasons for thinking that it is so. The hypoglossal nerve is often affected, and some cases of ophthalmoplegia are complicated with progressive muscular atrophy due to chronic anterior poliomyelitis. Apart from this, however, the cord is often the seat of disease, the posterior columns being most often affected; but sometimes it is the lateral or even the anterior columns which are altered.

Section VI. is the last, and deals with certain clinical considerations. Nystagmus is often present, and usually some mental affection, such as that seen in general paralysis. The clinical observer will also frequently detect symptoms of tabes dorsalis, multiple sclerosis, or affection of the posterior and lateral columns of the cord. But ophthalmoplegia may occur alone, and in any case ophthalmoplegia interna is not characteristic of tabes dorsalis. Occasionally ophthalmoplegia gets well, and if one can rely on Bristowe's and Eisenlohr's cases, it is sometimes a functional affection. It is not due to syphilis alone, though this disease seems to have been the cause in some cases. Sometimes the development of paralysis is very slow, and the loss of power

may remain confined to the muscles of the eye for as much as five-and-twenty years. But even after a long period of time other paralytic phenomena may appear.

SEYMOUR J. SHARKEY.

Psychologie de l'Idiot et de l'Imbécile. Par le Dr. PAUL SOLLIER. With twelve plates. Paris: Felix Alcan, Boulevard St. Germain. 1891.

THIS book is one of a series of volumes of "Contemporary Philosophy," and is an attempt by the author to elucidate the morbid psychology of idiots and imbeciles. A short paper on the subject by Dr. Bucknill appeared in 1866 in the eleventh volume of the 'Journal of Mental Science,' but this book contains no less than 276 pages. The author is well qualified to undertake the work, as he was formerly one of Dr. Bourneville's assistants at the Bicêtre, where there is a school for idiots and imbeciles, and he is now curator of the museum in that institution. His object, he says in his preface, has not been to show the more or less frequency of some of their psychical peculiarities, but to make a study of them all. He describes the sensations, instincts and sentiments which are known to exist, and examines fully the amount of intelligence, will, and responsibility which idiots and imbeciles possess. He confines his research to individuals who are young in age, partly, because his field of observation has been limited to these, but chiefly, because the period of youth is the most interesting time in which to study the evolution of the different faculties. He mentions the different definitions which have been given by authors and maintains that the faculty of attention serves as the best basis of classification. This opinion he defends at some length. Intelligence, according to Ferrier, is proportionate to the development of attention and to the development of the frontal lobes, and those of us who are engaged in the education of idiots are aware that the frontal lobes are often defectively developed in those whose power of attention is very feeble. Perez has remarked that in young children as well as in young animals the most attentive are apparently those in whom nervous sensibility is well developed. Sensation is known to be defective in idiots, so that anatomy and physiology equally tend to show the impossibility of normal attention in these beings. To develop intelligence it is necessary to develop the senses and

the muscular movements, but when the relation of the latter to one another is altered, one can easily conceive the difficulty of developing the attention. The author then refers to the three periods which Ribot distinguishes in its formation and applies the knowledge thus gained in examining the development of this faculty in idiots and imbeciles. Attention is spontaneous and voluntary: the first is the primitive form; the second is the result of education. Imbeciles are almost as difficult to educate as idiots who are a little elevated in the intellectual scale; in the latter it is difficult to attract the attention, in the former it is impossible to maintain it.

With reference to the psychological evolution of these patients, there appears to be a time when no more progress is possible, and all that one can hope for is to maintain the results which have been acquired. This culminating point varies in different individuals, and is connected with the development of the various psychological functions. Sometimes one faculty will be arrested and not others. In idiots one can often see suspension of development of the intellect at six or seven years of age, while evolution of the sentiments and the senses will continue up to eighteen or twenty years. In imbeciles the senses, sentiments, and intelligence cease to progress almost at the same time, generally when the patients arrive at a state of puberty. When the faculties decrease there is progressive weakening of the will, the intelligence, sentiments, and sensations, but the devolution is not manifested in the same way in idiots and imbeciles. In the idiot, there is a long period during which the results acquired remain permanent, but when retrogression does take place it is generally very rapid, and affects both the physical condition and the moral faculty. In the imbecile this weakening of the faculties progresses more slowly, and in consequence of the inequality of their faculties it is often produced in a partial manner. Taken as a whole, the subject of the book is well thought out, though all the author's conclusions cannot be accepted.

FLETCHER BEACH.

Abstracts of British and Foreign Journals.

Chantre on the Mechanism of lid-closure after section of the Nerves of the Orbicularis. (*Thèse de Lyon*, 1890; and *Archives de Physiologie*, 1891. .

WHILST experimenting on dogs with a view to elucidating certain points connected with palpebral restoration in man, the author observes certain facts of physiological interest. These observations relate to modifications in eyelid-closure, following (either immediately or after a time) on section of the nerves supplying the orbicularis muscle.

When both internal and external branches are cut, the eyelids cannot be brought into contact. It was observed, however, that slight up and down movements of the upper lid were still possible. These movements are passive, and are due to the reflex retraction of the eyeball, and return to its normal position at each voluntary or involuntary effort to close the lids.

Hence, after paralysing section, there can still be an incomplete lubrefaction of the eyeball owing to its movements forwards and backwards which allow the eyelid to drop and rise slightly.

These phenomena might, at first sight, be mistaken for evidence of persistent motility. Though the latter remains in abeyance, movements of the eyelids reappear after a while through another mechanism. About six weeks after unilateral section the dog closes its eyes spontaneously. Still, on the paralysed side, it is noticed that the closure is effected chiefly through the raising of the lower eyelid, whilst the upper lid glides passively on the retracted eyeball. At the same time, the upper lip and all the facial tissues are raised upwards, and push the lower lid over the eye.

All these movements may be excited from the conjunctiva, and by calling forth an elevation of the lip by touching its vibris sac, the same phenomenon is produced.

On stroking the head of the dog the normal eye is closed without lip-movement; whilst on the other, the closure is effected by the mechanism just described.

These facts demonstrate the intervention of neighbouring

muscles in the act of lid closure ; and this intervention, at first voluntary and insufficient, becomes by education reflex and effectual.

Thus we have here an instance of a derivation of reflex phenomena after interruption of their habitual circuit ; and of an increase of excitability in a centre normally uninterested in the performance of a certain movement.

In the animals so operated, the sensory stimulus from the conjunctiva being no longer reflected along the upper branch of the facial, is carried on to the lower branch ; the eyelids are then closed through the action of the facial muscles and retraction of the eyeball.

Finally, excitation of the infra-orbital nerve endings on the operated side, produce reflex facial movements, and thereby closure of the eye on the same side only.

The author's conclusions from his experiments are :—

(1) Immediately after section of the nerve to the orbicularis in the dog, the upper lid shows passive up and down movements, due to reflex retraction of the eyeball.

(2) About ten weeks after section, palpebral closure can be effected through powerful facial contractions (in addition to the effect of retraction).

(3) This intervention of the facial muscles is determined by a derivation of the conjunctival reflex upon the inferior facial branch.

(4) This modification in the direction of the reflex is accompanied with increased excitability of the centre calling out this new function.

PROF. ARLOING (Lyons).

Inglis on Friedrich's Ataxia and its relation to the Conducting Paths in the Spinal Cord.—At the Congress of American Physicians and Surgeons, Dr. David Inglis, of Detroit, read a paper upon the above subject before the American Neurological Association.

He reports in brief a case of Friedrich's ataxia in a boy of six years of age, in which the symptoms conformed accurately to Friedrich's own summary of the characters of the disease, viz. : " Impairment in the combination and harmony of movements developing gradually and spreading from the lower to the upper half of the body, and always involving finally the organs of speech. Sensibility and the functions of the special senses and of the brain being intact ; paralysis of the sphincters

and trophic disturbances are absent ; less common phenomena are curvature of the spine, sensations of vertigo and nystagmus. From a clinical point of view we must regard the disease as a progressive paralysis of the faculty of combination of movements."

A review of the thirteen recorded autopsies shows a practical agreement that the pathological condition underlying the disease consists in a progressive sclerosis which always affects the column of Goll, the column of Burdach also, but not so completely, the direct cerebellar tracts with Clarke's column in most cases, and the crossed pyramidal tract in some cases, but the sclerosis is here not so intense. We have to deal with a disease of the tracts which degenerate upward, which are usually looked upon as centripetal and as conveying sensory impulses.

The author contends that the symptoms of Friedrich's Ataxia afford a demonstration that these tracts do not convey sensory impulses upward, for sensation is not impaired, but that they are the main tracts for the conveyance of co-ordinated motor impulses downwards ; that their anatomical relations with the medulla, cerebellum and mid-brain, as well as the facts of Friedrich's disease agree in showing them to act to co-ordinate motor impulses of the mid-brain, cerebellum, and higher and lower levels of the cord.

The facts of embryology strengthen this theory ; at the end of the foetal life, at a time when the pyramidal tracts are undeveloped, the posterior columns and direct cerebellar tracts are complete. Their function evidently begins at once after birth. When we remember that the new-born infant is characterised, not by voluntary control of its muscles, not by accuracy of sense perception, but by an extensive co-ordination of involuntary motor functions, the conclusion is easy, that these, the only tracts fully developed at birth, subserve these purposes.

The direction of Wallerian degeneration is not necessarily the same as the direction of normal physiological impulses in any given nerve tract.

(AUTHOR'S ABSTRACT.)

BRAIN.

PART II., 1892.

Original Articles.

ON THE MECHANISM OF BRAIN INJURIES.*

BY ALEXANDER MILES, M.D., F.R.C.S. EDIN.

Syme Surgical Fellow.

[From the Laboratory of the Royal College of Physicians, Edinburgh.]

PRELIMINARY CONSIDERATIONS. VARIOUS THEORIES OF CONCUSSION.

THE question as to whether or not it is possible to have fatal injury to the head, in which there is no appreciable gross lesion to the brain substance directly, or through its membranes, so called concussion, has long been discussed by writers on this subject.

By the older authors, the celebrated case, recorded so graphically by Littré (1†) now nearly two centuries ago, was accepted as decisive of the possibility of such a thing, but being almost solitary, it was by some looked upon as the exception which proved the rule. Although the case most quoted, however, Littré's was not the only one of alleged fatal concussion, Sabatier (2), Boyn (3) and Mourier (4), have all left records of similar cases. In a more sceptical age these classic records have been somewhat severely criticised, and the light of a more advanced physiology and pathology, with the information derived from more carefully

* This paper formed part of a Thesis, presented for the Degree of Doctor of Medicine in the University of Edinburgh. A Gold Medal and the Syme Surgical Fellowship were awarded it by the Medical Faculty. Part of it was read before the Royal Medical Society in November, 1891.

† These figures refer to the corresponding numbers in the Bibliography at the end of the paper.

conducted *post-mortem* examinations, has thrown serious doubt on the accuracy of the observations on which the conclusions were based.

Sir Prescott Hewett (5) leads the attack against the theory of Littré, based on his oft quoted case, and concludes that "it has still to be demonstrated that death can result from mere concussion." Holmes (7), Bryant (8), McEwen (9), and Savory (10) in this country, and Neudorfer (11), Nancréde (12) and Fano (13) abroad, are, in the main, with Hewett; and so far as my experience, based upon *post-mortem* examinations, as well as a large number of experimental observations goes, I must agree with them.

The school which supports the old view is neither so strong, in point of numbers, nor so powerfully armed with statistics as is that of its opponents; but it includes men whose opinion is of sufficient weight to give them the standing of an influential minority. Huguenin (14), Hutchinson (15), as well as some others, believe that death may result from concussion without gross lesion. Mr. Hutchinson admits that lesions exist, but holds that they are to be looked upon "as an index of the violence of the shake, and not as causes of death, nor perhaps even as serious complications;" and Mr. Savory, while denying the occurrence of death without lesions, admits that "the effects must be due to the shock of the violence rather than to the lesions."

This raises the question, What is the ultimate cause of the symptoms in so-called cases of concussion of the brain? Is it, for example, as the old writers supposed, that the brain is thrown into a state of vibration which causes for the time being a suspension of its functions, as was held by Bruns (17), Liston (18) and Miller (19), on more or less purely theoretical grounds; or are the multiple capillary hæmorrhages on which Fano (13), Nélaton (21) and Rokitansky (22) laid such stress, the important causal pathological factors; or again, must we look to some profound vascular disturbance in the nervous centres as the "*fons et origo mali*?"

The Vibration Theory as stated by the late Professor Miller of Edinburgh, is as follows:—"Under the impulse of a blow or fall the brain must sustain more or less vibration if the cranium

remain entire. It is a pulpy organ, which exactly fills a nearly spherical bony cavity, whose parietes are elastic in a considerable, though very variable, degree; and if these parietes sustain any sudden change of shape, their contents must sustain a corresponding amount of compression, as any alteration in the shape of a spherical cavity must lessen its capacity. Whenever any external force impinges on the cranium with sufficient violence, it must be flattened at the point of impact, and expanded in some other direction; but these changes are, in virtue of the very cause whence they originate, of but momentary duration; the point primarily flattened by the compressing force immediately resumes its original shape, which is necessarily followed by a corresponding return of the expanded portion of the cranium to its previous dimensions. These oscillations may occur several times in rapid succession, their number and extent depending on the elasticity of the cranium, and direction of the force applied. In concussion then, the entire brain sustains a series of vibrations and momentary compressions varying in number and amount in every imaginable degree in different cases."

These views are supported by Bruns, who says that: "the vibrations permeate the brain as a whole, as well as its individual component parts." Gama (23) and Alquié believed that while the brain elements remained unaffected, the brain itself might be knocked from side to side of the cranium; but the results of these observers must be received with due caution, as their investigations were carried out on an artificially constructed apparatus, which could only imperfectly imitate the exceedingly delicate anatomical arrangements of the human brain.

There are several points in the vibration theory, thus enunciated, to which exception must be taken.

In the first place, the brain in its normal state, whether or not it be a "pulpy organ," does not exactly fill the cavity. There is everywhere a slight, and in some places a considerable space between it and the bone, filled by the cerebrospinal fluid, a fact of great pathological as well as anatomical importance.

Nor is the cranium, I submit, by any means a "spherical cavity." It is so far removed from a sphere, that the laws of physics which apply to cavities of that shape do not affect it. It has been shown over and over again that the capacity

of the cranium is in constant proportion to its size, any increase in the quantity of one element, *e.g.*, blood, being compensated for by an escape of another, *e.g.*, cerebro-spinal fluid, and *vice versa*. And it is by an escape of one or other, or both of these fluids that a depression of the parietes is able to diminish the contents of the skull, which, by the mere fact of being depressed, has had its size diminished, and therefore proportionately its capacity.

That this depression is not permitted by any appreciable diminution in size of the brain itself, is shown by the fact that it requires the pressure of a whole atmosphere to reduce a body of the consistence of the brain by $\frac{1}{25000}$ th of its volume, and in very few head injuries is the pressure anything like so great, Duret (24). This theory also leaves out of account the fact that the hemispheres are hollowed out by the ventricles, and that before pressure could directly diminish the volume of the brain substance these cavities would have to be obliterated.

Other anatomical arrangements in the shape of the falx cerebri and tentorium cerebelli, etc., seem to me so to fix the brain in position that any considerable displacement of it would be evidenced by lacerations, due to its impinging on these tight resistant bands.

Taking all these points into consideration, I do not think there is sufficient evidence to show that the vibration theory is tenable.

The next theory, namely, that the symptoms of concussion are to be accounted for by the multitudes of capillary hæmorrhages so often found in the cerebral substance, has as its chief supporters, MM. Fano and Nélaton, and with them in the main Stromeyer (25) agrees, although he differs from them as to when these extravasations take place, the former assigning them to the primary stage, the latter to the stage of reaction. Chassaignac and Sanson are also disposed to look upon these lesions as sufficient to account even for death in fatal cases.

Drs. Bright (20) and Blandin (16) do not go so far. They believe that "in addition to minute extravasations, patches of contusion of the cerebral substance are necessary to account for death, if not for less serious symptoms."

On the other hand Huguenin (14) and McEwen (9) do not believe that these scattered lesions are sufficient to account for the phenomena, and with them M. Duret (24) is at one. He says that he "sought as carefully as possible at the autopsies and found only small lesions quite insufficient to explain so grave phenomena."

Mr. Jonathan Hutchinson argues that these lesions cannot be the cause of the symptoms and of death, because many cases with worse lesions, not only live but have no symptoms.

Unfortunately for this theory multiple hæmorrhages are by no means constant, many cases occurring in which none are to be found. Besides, the fact that the symptoms of concussion, in many cases, so soon pass off, argues against their being due to lesions, which from their very nature, could only disappear after a period, at least much longer than is required for the cessation of all evidences of concussion.

Neither of the theories just considered, namely (1) that of physical mechanical vibration; and (2) multiple petechial hæmorrhages seems to be sufficient to account for the varied and general symptoms of concussion.

Is that of derangement of the cerebral circulation any more acceptable?

When we consider the general character of the symptoms, how widely spread they are, affecting every function and organ in the body, from the prefrontal lobes with their highly specialised actions, to the least important and simplest mechanical muscle, we comprehend how almost impossible it would be for any single focal lesion, however diffuse, to be held responsible for all of these. And if we bear in mind the close resemblance which this group of symptoms bears to other conditions—such as cardiac syncope, reflex syncope from pain, or severe mental shock, where the cause is acknowledged to be a disturbance of the circulation of the brain—we are induced to accept this explanation of the phenomena, as at least a more workable theory than either of the other two. Most modern writers accept this view of the subject.

Some, such as Savory (10), McEwen (9), Le Gros Clark

(6), and others, define concussion as a form of "shock," which is only modified in so far as the hurt has expended its force on the brain.

Mr. Hutchinson (15) regards "anæmia of the brain" as being "the proximate cause of the phenomena of concussion," but whether this condition is produced by contraction of the arteries, or by diminution of the venous tension leading to congestion, and so an "oxygenless" condition of the cortex, he does not give an opinion.

Huguenin (14) accounts for the anæmia by the blood being squeezed out of the vessels by momentary compression of the brain substance, whose normal function depends on a proper supply of oxygenated blood, and which so suffers from thus being temporarily deprived of it, that its functions are suspended for a period varying in proportion to the severity of the injury as indicated by the amount of vascular disturbance.

It is doubtful if simple mechanical compression of the brain substance is the cause of the anæmia, as we have already seen that a much greater force is necessary to accomplish this than is almost ever applied in simple cases of concussion, and Nothnagel (28) has tried to explain the contraction of the blood-vessels on the assumption that it is the reflex effect of strong irritation of peripheral nerves. He does not explain, however, how this state of contraction can be kept up reflexly for several days, as is sometimes the case in concussion. Jolly (26), Rigel (27), and Frey (29) have failed to reproduce these symptoms by reflex irritation of vaso-motor nerves, although Goltz has produced a reflex paralysis of the vessels by injury to the head, which supports Fischer in his belief that this is the true explanation of the symptoms.

I propose now to consider what clinical and experimental proof we have of the existence of such a vascular disturbance.

INFLUENCE OF CONCUSSION ON THE HEART AND GENERAL CIRCULATION.

The experiments and observations on animals to which I am about to refer are described in detail in another part of

my paper. Unless the injury was intended to kill the animal instantaneously, full chloroform anæsthesia was produced before the experiment was commenced.

In the first case, that of a rabbit before the injury was inflicted, when the animal was in an excited state, the pulse was 200, and only countable with difficulty. After a severe occipital blow had been delivered however, there was marked slowing of the pulse. During the first minute following the blow no cardiac impulse could be felt whatever. During the second minute the action was very irregular—for the first fifteen seconds the pulsations were at the rate of forty to the minute, but this was followed by a rapid succession of small beats which were quite uncountable, but which, if counted, would of course have raised the number of beats per minute considerably. This irregularity, however, was evidently due to the action of respiration, at all events, it was synchronous with long-drawn, deep inspirations, and if we assume the causal relationship between the two, then the heart *per se* was certainly slowed, and that in a marked degree, and whether we make this assumption or not we can safely say that the circulation was seriously interfered with.

In another experiment on a rabbit, before a moderately severe frontal blow had been administered the heart was beating regularly and at the rate of 190 per minute. During the first minute after the blow the heart was acting so tumultuously as to be quite uncountable, and continued to do so for the next two minutes. At the end of four minutes, however, it was again regular, vigorous, and acting at the rate of 130. The animal was deeply under chloroform.

In a third rabbit, from which the cerebro-spinal fluid was rapidly aspirated under chloroform, the heart was from the first slow and feeble.

In a fourth experiment where the pulse before the blow was given was 180, rapid and small, two minutes after, it was down to 120, and described as full and bounding.

M. Duret (24) has demonstrated in an experiment carried out with the object of ascertaining accurately the heart rate following a blow to the head, that there is a

period during which the pulse is very slow, full and bounding, and this period he finds is synchronous with a disturbance and slowing of the respiration.

The experiment was performed on a dog with the following results :—

Before the blow the pulse was	100 p. min.
1st minute after blow	14 „
2nd „	„	...	16 „
3rd „	„	...	12 „
4th „	„	...	12 „

This stage is followed by another, during which the pulse, while becoming faster, is of very low tension, even dichrotous (“*avec gruppetti*”) and this stage corresponds to an acceleration of respiration.

6th minute after blow pulse was	...	68 p. min.
7th „	„	68 „
20th „	„	40 „
30th „	„	54 „
2 hours „	„	60 „
4 „	„	93 „
8 „	„	82 „

This observation seems to agree for the most part with what I have gathered from the results of my experiments, which I may here say were not performed to determine this point particularly, and so are less detailed than they would have been otherwise. The conclusion these observations warrants is, that following a severe blow to the head there is usually a certain degree of slowing of the heart, and in all cases a profound disturbance of the general vascular system, a disturbance which must seriously affect the organs of the central nervous system.

DOES THE BRAIN BECOME ANÆMIC OR HYPERÆMIC ?

It is well known from the researches of Vulpian (30), Claude Bernard and others, that a reflex vascular spasm may be produced by irritation of sensory nerves of the body, or by stimulation of the restiform bodies, thus raising the general arterial blood tension, and that this increased tension is due to the contraction of the peripheral vessels

with accumulation of blood in the larger ones, and increased resistance to the heart's action. That such a spasm of the vessels of the brain exists, was demonstrated by Beck, who showed that under such conditions the surface of the brain was pale, and the ordinary pulsations invisible. Duret has confirmed this observation, and adds that the vascular spasm increases in proportion with the amount of violence used. Roy and Sherrington (47), however, state that direct stimulation of the medulla causes congestion of the brain, the vessels passively dilating, but they introduce a fallacy into their experiments by providing for a free escape of the cerebro-spinal fluid, so depriving the vessels of what support they receive from that fluid, and leading to their dilatation.

I hope to be able to show later, that in head injuries there is such an irritation of the restiform bodies as would account for the origin of such a reflex action. Meanwhile let me remark that the result of this alteration in the vascular supply of the brain, whether by spasmodic contraction of the vessels, or by their passive dilatation, will be to deprive the nervous substance, for a time at least, of the copious supply of pure oxygenated blood which is so necessary for the perfect performance of its functions; in other words it produces a temporary anæmia of the brain, just as would be done by bleeding an animal suddenly and almost completely, or by injecting lycopodium seeds into its vessels.

Kussmaul and Tenner (51) made some experiments on the effects of bleeding animals, and in these they produced symptoms which very closely resemble those observed in a number of my experiments, where very severe blows were dealt to the head, such symptoms as tetanism, marked changes in the pupils, localised convulsions, and so on, being very common.

In support of the contention that in my cases the phenomena were due to cerebral anæmia, just as they were in the animals which were bled, I may quote the conclusion come to by these observers, after they had produced similar symptoms by cutting off the blood supply to the brain. They say "that the interruption of the conveyance of arterial

blood to the brain produces tetanic symptoms as surely as hæmorrhage does."

Sir Astley Cooper (52) demonstrated the same point when he tied both carotids and vertebrals in a dog, with the result that the animal "lost sensation, had great respiratory trouble, with dilated pupils, it fell on its side and became violently convulsed. Stupor and general paralysis lasted for two days, and then gradually passed off, the animal recovering completely."

The series of symptoms produced in this animal, and undoubtedly due to cerebral anæmia, corresponds so closely to what I have again and again seen in my experiments, that I feel considerable confidence in believing, especially when supported by the facts brought out by Vulpian and Bernard, that the pathological condition in both is essentially the same, the only difference being in the originating cause of the anæmia.

To sum up then, I think the weight of evidence is in favour of the view that the symptoms of cerebral concussion are due to a profound disturbance of the circulation of the brain, and that the alteration is in the direction of anæmia.

ON THE EVIDENCE IN FAVOUR OF STIMULATION OF THE CORPORA RESTIFORMES BEING THE CAUSE OF THE VASCULAR DISTURBANCE IN CEREBRAL TRAUMATISM.

While making observations on a series of cases in which rabbits, pigs and birds had been subjected to blows on the head, varying in severity and in the exact region to which they were applied, I was struck with the similarity in the symptoms which immediately followed such injuries.

Let us take the case of a rabbit to which a severe blow had been dealt, as an example.

In the first place, the animal deeply anæsthetised, was thrown into a violent tetanic state, with greatly exaggerated motor phenomena, these lasting only a few seconds, and being followed by other motor symptoms less violent and more localised, such indeed as might be attributed to some lesion of a particular part of the brain. These effects I have

observed in a great many of my experiments, in fact, in all in which the blow has been of even moderate severity.

As time wore on the phenomena of this stage, which Duret has appropriately called the "tetanic stage," were gradually replaced by others of a more serious nature. The rigidity and jerkings of the limbs gave place to paralysis, the animal could no longer be roused, and the respiratory and cardiac functions were so interfered with as to cause death.

The symptoms of the tetanic period are illustrated by the following experiments—in all of which, save those on pigs, the animals were fully anæsthetised before the blow was delivered, so that the element of pain does not require to be considered. The pigs suffered from swine fever and were consequently being killed at the Edinburgh slaughter house, where I had the opportunity of observing them.

(1) An occipital blow delivered to a rabbit, was immediately followed by spasmodic convulsions of all the muscles of the body; and these by violent seizures which caused the animal to rotate round its long axis.

(2) A severe blow over the frontal region of a young pig caused the animal to fall. It then lay on its back for a few seconds, all its limbs, but especially the hind ones, being violently moved. There was no distinct opisthotonos.

(3) In another pig a blow of moderate severity was dealt over the vertex. With a scream the animal passed into a state of extreme opisthotonos; for an instant it was in a state of extreme rigidity, and then all its limbs became violently agitated.

(4) In this case the blow was delivered over the left parietal region of a rabbit, while the creature was deeply anæsthetised, and rigidity of all the limbs resulted at once.

(5) In this case the blow was over the occiput, the subject being a young domestic cock, and the result was the same; the creature passing into a condition of extreme tetanus, followed for a few seconds by jerking of the limbs.

These examples will suffice to show that in cases of severe head injury in animals of different genera and even classes, and applied to different parts of the head, practically the same results are produced in the first few seconds after the hurt has been sustained.

And if we look at the gross lesions found in these cases after death, we fail to discover a satisfactory explanation of all the phenomena—opisthotonos, pleurosthotonos, agitation of all the limb muscles, sometimes spasms of the facial muscles, the ocular muscles, and even a combination of all these.

But even did we find a hæmorrhage or a laceration in some particular part of the brain, which could have been the cause of symptoms so serious and so widely diffused, we have still to face the difficulty that these symptoms follow so rapidly on the blow that they cannot be attributed to lesions, which, from their nature must of necessity take some little time to be produced.

It seems therefore that this consideration strengthens the view that the phenomena are due to a reflex action, as was first pointed out by Duret.

But for a reflex action we require an efferent stimulation. What then is the area stimulated in these cases? We know from experience that injuries to the scalp, even much more severe than those produced in the cases quoted, do not give rise to such symptoms; and it has been shown that direct stimulation of the nerves of the dura mater likewise fails to produce such a gross reflex manifestation.

We must therefore look to some part of the brain itself for an explanation, some part which has a particularly sensory function, and in which lesions are constant in such cases.

The notes of my observations shew that the area most frequently injured is the bulb, and it has been pointed out by Duret that the most sensitive part of the bulb is the restiform body, by irritating which *mechanically* he has succeeded in producing all the phenomena of this tetanic stage of concussion. He says that “the phenomena of the first stage of concussion find a very satisfactory explanation chiefly in the irritation of the restiform bodies. . . .” and this statement is supported by the fact that in all the cases just referred to there is evident enough source of irritation of these bodies, and that in those in which the symptoms were most marked the lesions are also most prominent.

The next points to be decided are—"How are these parts stimulated? How are the lesions so often found in the region of the bulb produced?"

ON THE ANATOMY AND PHYSIOLOGY OF CEREBRO-SPINAL FLUID.

The physiological importance of the cerebro-spinal fluid has been carefully studied by John Hilton (41), Richet (43), Salathé and others, who have shown that in all probability its function is to permit of the variations in volume of the brain which are known to accompany functional activity, great muscular or respiratory exertion and so on.

Now it is evident that the rigid cranium can take no part in providing for these variations in size, and it is equally evident that they must be provided for, because the delicately constructed cerebral tissue would not be tolerant of such pressure as would otherwise inevitably result, without exhibiting important symptoms. We would therefore conclude, and there is abundant experimental and clinical evidence in support of the view, that the cerebro-spinal fluid is the safety valve of the brain.

Believing that the cerebro-spinal fluid is the all-important factor in the production of the phenomena of concussion, and of the lesions found in severe head injuries, I must briefly consider the anatomical distribution of this fluid in the nerve centres.

A. Everywhere the brain is surrounded by a layer of cerebro-spinal fluid, varying in thickness or depth at different places, constituting what has been called the "water bed of the brain." The main "lakes" or "cisterns" are situated in the region of the base and also along the course of the larger blood vessels, such as the sylvian artery.

B. The brain is hollowed out by a series of cavities—the ventricles—and these are filled with cerebro-spinal fluid.

C. In connection with each blood vessel as it enters the brain substance there are two distinct lymph spaces:—(a) The adventitial lymph space of Robin, between the tunica muscularis and the tunica adventitia, and continuous with the sub-arachnoid space; and (b) the peri-vascular lymph channel of His, between the adventitia and the brain substance, and continuous with the epicerebral space. These spaces, the latter of which continues to surround the blood vessel on to its minutest ramifications are filled with cerebro-spinal fluid, in which the blood vessels are

suspended. This is the anatomical point previously overlooked in this relation.

D. And lastly, the nerve cells themselves are similarly placed, each cell being, as it were, suspended in a small excavation in the brain substance, called a pericellular sac. The communication between the perivascular spaces, and the pericellular sacs depends on the connective tissue cells of the neuroglia, which are distributed along the adventitia of the arterioles, and on to the arterio-venous capillary plexus, and thus establish a connection between the sacs and the spaces.

We are able to trace in successfully prepared sections, in close proximity to every nerve cell a branch of a capillary vessel, and it is to the sides of this loop that the pericellular sac is attached, the nerve cell being apparently suspended in the sac by means of its processes, which, passing through the free space of the sac proceed in all directions into the neuroglia. As these sacs are never terminal the nerve cell is bathed in a constantly renewed lymph bath.

In addition to these perivascular spaces and pericellular sacs, Bevan Lewis (37) describes a terminal set of lymphatic elements which consist "of a system of plasmatic cells with numerous prolongations, which are always in intimate connection with the adventitial lymph space, and which drain the areas between the vascular branches, these we have termed the lymph connective elements."

These four elements, all in direct anatomical continuity with one another, go to make up the cerebro-spinal fluid system of the brain.

Here I may remark that the fluid surrounding the brain is in direct continuity with that around the spinal cord, and that its transit from one cavity to the other is a comparatively easy matter; while the intra-ventricular and sub-arachnoid fluids communicate directly only through a very small aperture in the region of the fourth ventricle—the foramen of Magendie (35).

ON DURET'S THEORY OF CEREBRAL CONCUSSION.

The research which led M. Duret to formulate the cerebro-spinal fluid theory of concussion was carried out in the laboratory of M. Tillaux, and the result published in 1878.

In the first of his experiments he forcibly injected into

the cavity of the cranium of a dog, through a small trephine opening, about 300 grammes of water. After severe spasms of the limbs, opisthotonos, and cardiac and respiratory troubles, the animal speedily died, and the brain presented quite a series of very severe lesions, such as a rupture in the floor of the fourth ventricle, tears in the substance of the pons and medulla, hæmorrhages into the restiform bodies, third ventricle, and on to the surface of the brain.

Believing that this enormous destruction of brain tissue might be due to the increased pressure produced in the cranial cavity, and especially in the ventricles, by the large quantity of fluid so rapidly injected, he varied his next experiment by using a coagulable fluid, namely, gelatine, injecting it very forcibly between the dura and the skull. After exhibiting symptoms similar to the last animal, only somewhat more exaggerated, death followed in a few seconds. The lesions corresponded in a general way to those in the previous case, but were on the whole more severe.

These lesions M. Duret believes are all due to the sudden forcible pressing of the fluid of the lateral ventricles backwards through the aqueduct of Sylvius on to the fourth ventricle, and thence to the cord. The fluid in the lateral ventricles being five or six times more than the fourth ventricle can possibly contain, the latter cavity is subjected to such enormous pressure that its walls are torn.

On comparing these results with my own, produced by the less artificial means of administering blows to the surface of the skull, one cannot but observe the remarkable correspondence in the two series, not only as regards the symptoms shown, but also in the nature and distribution of the lesions observed after death.

It is, however, to be observed that in the injection experiments the symptoms and lesions are alike much more serious than in the percussion series—doubtless on account of the greater force employed, and the more direct way in which it is applied to the brain and its contained fluids.

But, however similar the results in the two cases may be, I cannot admit that the means of producing these are so nearly comparable as M. Duret would appear to indicate. In

the one case the force acting on the head is momentary in its duration, and the cranial contents could, but for any lesions produced by the blow, readily resume their normal conditions and continue to act as before. In the other series, however, the material injected necessarily remains, and so profoundly alters the physical and physiological conditions of the nervous centres that a return to their *status quo*, even supposing no lesion had been produced, would be absolutely impossible. In this, therefore, I think Duret's experiments are much better adapted to the elucidation of the symptoms and pathological appearances of *compression* than of *concussion*. At the same time, they have served to draw attention to the mechanism by which concussion symptoms and lesions—for I think we must admit there are lesions in concussion—are produced, and it is to the confirmation of these observations that I desire to direct my attention, by experiments performed more in accordance with the conditions under which our patients receive their injuries, and therefore more likely to lead to a useful clinical appreciation of the at present obscure symptoms, more obscure pathology, and still more obscure remote prognosis in cases of severe head injuries (48).

It is undoubtedly exceedingly difficult to distinguish clinically between Concussion, Compression and Contusion of the brain, and even after the most careful examination of the symptoms in an individual case, even experienced surgeons are unable to form a decided opinion. This is of course due to the fact that these pathological entities seldom exist singly—a blow which is serious enough to produce marked symptoms of concussion, is usually sufficient to cause gross lesion of the membranes, giving rise to hæmorrhage enough to cause symptoms of compression, which take the place of those of concussion as they tend to pass off. Or should the membranes and their vessels escape laceration, the nervous substance itself suffers injury enough to permit of blood being extravasated, and so raising difficulties in the diagnosis. M. Duret in speaking of this difficulty speaks of “doubt, which has not ceased after the autopsy.”

ON THE MECHANISM OF CRANIAL AND CEREBRAL INJURIES.

With these preliminary observations we may proceed to consider how severe blows and other injuries to the head produce their effects.

“Cone of Depression,” and “Cone of Bulging.”

It has been shown by Félizet (44) that the skull is elastic in a high degree, so much so, that it will rebound from the ground to a considerable extent if dropped from a height.

By smearing the vault of the skull with ink, and then dropping it from a height on to the floor, and comparing the size of the stain left with the area of rounded skull which could be in contact with the flat floor at one moment, he found that the former exceeded the latter in size. This could only be accounted for by the area around the point of contact being momentarily increased by the weight of the skull.

In another series of observations the skull was filled with soft paraffine, and then allowed to fall forcibly on to the ground, and it was found that a large depression was left on the paraffine, exceeding in size the possible area of contact, being distinctly rounded in outline and deeper in the centre than at the margins.

This circle of depression, or as M. Duret more accurately describes it, this “cone of depression,” represents the extent to which the cranial parieties are depressed short of being fractured.

In order to account for certain phenomena observed in cerebral traumatisms, Duret states that at the opposite pole of the axis of percussion, the vault of the cranium presents a corresponding “cone of bulging.” He does not, however, state whether he merely assumes this cone of bulging, or whether he has demonstrated it. If the latter, he does not describe the method by which he has done so, nor does he quote the experiments of others bearing on the point.

Experiments on Production of Cone of Bulging.

As I believe this to be a most important step in his line of argument, I have made a number of experiments to satisfy myself as to the accuracy of his statement.

These I did on the cadaver as soon after death as I could obtain an opportunity, so that the physical conditions might be as nearly similar to those existing during life as possible.

I obtained a quantity of glazier's putty, of such consistence that it could be handled without losing an impression made on it, and having oiled the skin to prevent too close adhesion, spread a pretty thick layer over one or other frontal region. I then marked out what I judged to be the opposite pole of an axis of percussion, and dealt a sharp blow over that spot with a wooden mallet.

In a majority of the cases a distinct impression was left on the putty. I varied the position in which I dealt the blows, with similar results.

In another set of experiments with the same object, I spread a smooth layer of vaseline about a line thick over the forehead and then dealt a smart blow over the opposite pole.

In some, although not in all cases, a clearly marked ring could be seen on the surface of the vaseline, standing out very much as butter stands out from bread when two surfaces have been separated. In one of the cases in which no such ring could be detected, the head had come off the mallet, and the force thereby misspent; in the other negative cases the only explanations I can suggest are (1) The blow not being strong enough to depress the skull at the point of contact, and (2) the blow being too diffuse to form a defined "cone of bulging."

The results, however, seem to confirm what Duret had assumed (or proved), and are, I think, of importance in so far as they help to explain the mechanism of lesions so often found at the opposite pole of the axis of percussion, in head injuries—that is, at the so-called point of *contre-coup*.

In view of these facts, I take it that the mechanism of

an injury to the head producing gross lesions, is, briefly, this :—

A blow is dealt to the cranial wall, and surrounding the point of impact the skull is depressed, forming the “cone of depression,” and from the area into which this cone projects the cerebro-spinal fluid is forcibly expressed. Where does this fluid go? We have seen that under such conditions, at the opposite pole of the axis of percussion, there is formed a “cone of bulging,” which would accommodate all or most of this displaced fluid. What does not find accommodation there will either pass along the perivascular lymph spaces into the brain substance, along the nerve sheaths, or into the subarachnoid space of the cord.

The cones of depression and bulging, however, are only of momentary existence, the elasticity of the skull permitting it at once to return to its *status quo*. Therefore, at each point there is formed a vacuum, first at the cone of depression when the bone recoils, and next at the cone of bulging when the fluid recedes. The result of this is, that the blood-vessels of the membranes and brain substance at these points are left for the moment unsupported, and they rupture, a result which is favoured by the fact that the compression to which they have been subjected has partially paralysed them.

For this rupture of vessels suddenly deprived of support derived from fluid under tension we have several surgical analogies—notably where, after withdrawing all the fluid from an over-distended bladder, we have hæmorrhage from the vessels of the lining membrane, or it may be even from the kidney, if the backward pressure has been sufficient to affect that organ.

Such a mechanism would explain the occurrence of lesions, not only at the seat of injury, but at the opposite point of the skull and brain, the so-called lesions of *contre-coup*.

ON “CONTRE-COUP.”

That this term *contre-coup* has become a most useful one clinically there is no doubt, but it is by no means so certain

that it has as satisfactory a pathological significance ; and it is still more doubtful if those by whom it is used always entertain any very definite idea or theory as to what it means.

Mr. Erichsen (45) says that "the explanation . . . is, that the blow starts a wave in the soft cerebral substance, which breaks against the bone on the other side. In very rare cases it is possible to trace the course of this wave by hæmorrhages in the cerebral substance in its track." We have already seen that Gama (23) and Alquié deny that the brain elements can be thrown into vibrations by a force applied to the surface of the skull.

Le Gros Clark (6) denies the admissibility of the term *contre-coup* in the physics of the cranium at all. He points out with some considerable show of reason, that while a series of vibrations set up at a particular point in the circumference of a perfectly spherical glass globe, might find a point of convergence opposite the point of impact, such a result is physically impossible in the skull. When we consider its irregular shape, its varying thickness, the projections externally and internally, the numerous muscular attachments on its outer aspect, the adhesion of the dura mater and its processes on its inner, and the presence of the cerebro-spinal fluid—all of which would either absorb or transmit in different directions the vibrations set up, we see how exceedingly improbable it is that these vibrations would ever find a point of convergence, and especially one situated exactly at the opposite pole of the axis of percussion.

Although we are not here specially concerned with the question of fracture of the skull from *contre-coup*, it bears so close a relation to what we have just considered, that we may glance at it in passing. That it cannot be due to vibrations finding a focal point at the seat of fracture is, I think, beyond doubt, and it is scarcely more likely that the force of the soft resilient brain impinging on the hard, elastic skull could cause fracture of the latter.

In spite of the strong opinion of Mr. Erichsen, who "cannot doubt it," as he has "seen unequivocal instances," I think Mr. Le Gros Clark is nearer the truth when he says

that "a blow on one side of the head could only produce a fracture on the other side, if the head were knocked against an unyielding object," and in a great majority of cases of severe head injury the patient does fall, usually in the direction of the blow, thus providing an explanation such as Clark suggests.

While I believe that most of the so-called fractures by *contre-coup* are produced in this way, I think that in the light of the experiments on the "cone of bulging," others may be explained on the theory that this cone has gone beyond a point from which it could recoil and the bone has given way. After breaking, the skull might resume its previous level and the only evidence be a fissure; or, as in the rare cases quoted by Mr. Walsham (46), the fractured bone raised above the level of the skull constitutes what is called an "elevated fracture." A specimen of this condition is in the Museum of St. Bartholomew's Hospital, No. 879a.

Fracture of the base by a fall on the vertex is often referred to as an example of genuine fracture by *contre-coup*, but as Le Gros Clark points out, this is really a fracture by direct violence, the weight of the body being transmitted through the spinal column, and impinging on the occipital condyles. This view is borne out by the fact that very often the basal fracture is much greater than that of the vault, being therefore presumably the primary lesion, and in addition the fracture of the vault often appears to be spread from the base.

If we accept this theory of *contre-coup* lesions—namely, that they are due to the displacement of the cerebro-spinal fluid by the cone of depression, and its sudden recession from the cone of bulging leading to rupture of blood vessels, and consequent contusion and laceration of brain substance,—it will be evident that these lesions will vary in position according to the point struck.

If the frontal region or vertex be the seat of the cone of depression, it will be at the base of the skull that we must look for the evidences of the cone of bulging. We can scarcely expect, however, that any ordinary violence will cause an outward bulging of the body of the sphenoid bone,

and we have already seen that when it is fractured it is not from violence applied to the vertex, but to the super-incumbent weight of the body. The increased quantity of fluid therefore must swell the volume of the cerebro-spinal lakes at the base, and thus the force will be transmitted to the nerve elements themselves. That all fatal cases of concussion are associated with peribulbar lesions was stated by M. Fano in 1853, and with him in a general way Duret agrees. The results of my own experiments, which will be detailed later, have been to confirm these observations.

The late Mr. Vincent, quoted by Spence (47), evidently appreciated this element of the transmission of blows to the base of the brain, because he states as his opinion, that the amount of danger in cases of concussion depends largely on the direction, as well as the degree of the force, being greatest when in a line directed towards the base of the brain.

Mr. Bryant (48) also, by clinical observations, bears out the opinions of the various experimenters named, when he says that "in almost all cases of severe concussion the base of the brain is injured."

EXPERIMENTAL EVIDENCE IN FAVOUR OF CEREBRO-SPINAL FLUID WAVE THEORY.

After a blow to the head, in addition to the wave set up in the cerebro-spinal fluid external to the brain, *i.e.*, in the sub-arachnoid space, there is another and perhaps a more important disturbance, in the intra-ventricular fluid.

When the cortical substance of the hemispheres is pressed upon, it finds in the ventricles and their contents a sort of buffer which enables it to withstand shocks in a manner which would be impossible were each hemisphere a solid mass of brain substance.

Now, let us suppose the head is the subject of a severe blow, say over the frontal bone, a cone of depression is formed, which displaces the sub-arachnoid fluid in this region, and by the mechanism which we have already considered, a laceration of the brain or its membranes may take place. Not only so, but the force passing through the frontal lobe sets the fluid contained in the lateral ventricles into motion

in the direction of the axis of percussion, *i.e.*, towards the base of the brain. Here, however, there is considerable difficulty, because the only point of escapement is through the narrow aqueduct of Sylvius, and through this the fluid rushes, the effect on the aqueduct and its boundaries varying with the force employed, and its direction. When the force is great, the aqueduct may be torn up and lacerated, while in less severe cases it is only stretched. Whichever condition exists, after travelling the narrow aqueduct the fluid impinges with increased force on the wider fourth ventricle, which, as Duret puts it, "forms a cone of bulging ready prepared," with the result that the pressure on this most important locality is enormously increased, not only by the surplus quantity of fluid thrown in, but by the difficulty with which it can escape, there being only the minute central canal of the cord, and the scarcely larger foramen of Magendie to provide for the overflow. Under such circumstances we are not astonished to find primary lacerations of these delicate structures.

We have seen that the lesions on the surface of the hemispheres are in all probability due rather to the aspiration of the receding cerebro-spinal fluid, than to the pressure of the impinging wave, and I believe it is so too, in the ventricles and at the bulb. It is not uncommon to find, as I shall be able to show from my experiments, that the lateral and fourth ventricles are the seat of extensive extravasations, due, I consider, to the rupture of the unsupported vessels of the choroid plexuses during the ebb of the fluid wave.

In order to determine if the absence of the cerebro-spinal fluid would lead to rupture of these vessels and consequent hæmorrhage, I made several experiments by aspirating it.

These I shall proceed to describe.

Experiment 1, Rabbit H.—To determine the effect of rapid aspiration of cerebro-spinal fluid on the vessels of the brain substance, especially those of choroid plexuses.

November 21st, 1890, noon.—The rabbit, an adult male of average size, and in good condition, having been brought thoroughly under chloroform, an opening was made through the occipito-

atloid ligament, and the finest needle of an aspirator passed in. The trocar was then withdrawn, the cannula connected with the vacuum, and the cerebro-spinal fluid *rapidly* aspirated. Between two and three drachms were removed. During the aspiration the animal once had several slight spasms of the facial and limb muscles. These were so slight that at the time they were regarded as probably simply contractions of the panniculus muscle, and that they were due to reflex irritation of the left restiform body, which might have been irritated by the point of the needle, which was turned to that side after the membrane was perforated, and was felt to touch something. The cannula being withdrawn, the puncture was covered with collodion and wool. Antiseptics were used at the operation.

After aspiration the animal lay on its right side evidently in a perfectly unconscious condition. Pulse feeble and slow, breathing very shallow and rapid. No other sign of life was given. Loud sounds made close to the ears did not startle the creature.

Notes.—This moribund appearance has continued for an hour. General condition now improving. Pulse better, respiration normal. Both pupils dilated equally. The muscles of the right side of the neck are tonically contracted, forcibly bending the head to that side. All the muscles of the limbs seem to share in this tonic rigidity, though to a less extent than those of the neck. On the other hand, the muscles of the trunk appear to be paralysed, or at least in a state of paresis, because the animal is quite unable to move its body from the ground, although it can move about. In doing so, it exhibits an amount of inco-ordination of the limb movements. It now gives evidence of seeing, and of hearing loud sounds. Died soon after.

Autopsy.—As in life head forcibly turned to the right and dorsiflexed.

Brain.—Hemispheres look pink and congested; vessels on left side, both above and below, distinctly more engorged and prominent than those on right side. No hæmorrhage on surface.

Cerebellum congested in same way as cerebrum. Just over the cerebellum, in the space between the posterior ends of the two hemispheres is a small blood clot about size of split pea.

Interior of Brain.—On opening up hemispheres large clot found filling up the left lateral ventricle, and resting on upper aspect of basal ganglia and corpora quadrigemina of that side. The small clot seen above the cerebellum is found to be the termination of this one. Similar clot but much smaller found in right lateral ventricle.

Bulb.—Over left side of medulla in region of superior olive and restiform body is a small laceration, probably caused by the needle in aspirating. Near lower end of medulla, towards middle line and extending to left side is a hæmorrhage into the substance of the part. It extends along the medulla for about one-eighth of an inch, is irregularly rounded in shape, and has a piece of apparently normal brain tissue in the centre.

The aqueduct of Sylvius is dilated and exhibits one or two small hæmorrhages round it, the largest being in the left side of the corpora quadrigemina.

Cord is very pale and anæmic.

Remarks.—This experiment appears to bear out the theory that the sudden withdrawal of the cerebro-spinal fluid, and the consequent leaving of the vessels unsupported, leads to rupture of the latter and extravasation of blood into the brain substance (*e.g.*, the medullary hæmorrhage) and into the ventricular cavities. It also shows that the rushing of fluid through the aqueduct of Sylvius not only leads to dilatation of that passage but also to hæmorrhage into the comparatively dense tissues which surround it. Of course it may be suggested that the hæmorrhages were due to the aspirating force acting on the blood vessels after all the fluid had been withdrawn. I tried to guard against this fallacy by limiting the power of the vacuum, only partially exhausting the bottle. Besides, one's clinical experience in the use of the aspirator does not give much support to this view.

The symptoms due to the actual withdrawal of the fluid were neither numerous nor important, the animal of course being deeply anæsthetised. I have already said that the slight spasms, if such they could be called, were probably reflex.

The coma, rigidity of the limbs, with deviation of the head to one side, and slight variation in these irritative indications, are to be accounted for by the hæmorrhage into the left lateral ventricle, though why this hæmorrhage should have been more abundant on one side is not so easy to understand, unless, indeed, it be that the turning of the needle point to that side determined the direction of the flow. This we would not expect, because the needle should

have been in the inferior cerebellar lake, and the fluid to reach it would have to traverse the foramen of Magendie, where any inequality in the streams from the right and left ventricles would be equalised. The fluid of course may have escaped from the lateral opening of Key and Retzius. However, working in such a limited space and with such small quantities of fluid, the sources of error are so numerous, that we must not draw too wide conclusions from a single case.

To the hæmorrhages in the corpora quadrigemina we may assign but few symptoms. Abnormalities in the action of the ocular muscles often depend on lesions of these bodies, but I think the general comatose condition of the animal is amply sufficient to account for the phenomena in this case.

The peculiar inco-ordination of movement is explicable on one of two theories. We know that in certain cases of tumour of these bodies this is a prominent symptom, but Dr. Gowers (49) has pointed out that it is probably due to the pressure on the middle lobe of the cerebellum, rather than to any influence on the corpora quadrigemina. Here we had both hæmorrhage into the corpora quadrigemina and pressure, though slight, on the middle lobe of the cerebellum by the posterior end of the intra-ventricular clot. Probably both played a part.

The condition of the pulse and respiration most likely depended on the large hæmorrhage into the substance of the medulla, caused I believe by the recession of the fluid supporting the blood vessels.

The excessive congestion of the vessels of the left hemisphere, doubtless was due to the increased difficulty in the return of the venous blood, on account of the large clot blocking the vessels of the choroid plexus, and secondarily all the vascular arrangements of that side.

So far as it goes, I think this experiment is satisfactory in showing that by rapidly withdrawing the cerebro-spinal fluid, hæmorrhage can be produced in the cavities and substance of the brain.

The next points to determine are, what is the effect of

slow aspiration of the fluid; and what is the result of a severe blow on the head when the cavity has been deprived of its cerebro-spinal fluid.

The following experiment fortunately, throws light on both of these points.

Experiment 2, Rabbit I.—To determine the rôle of the cerebro-spinal fluid in the production of lesions from external injuries to the head.

November 21st, 1890, 1 p.m.—The subject of the experiment was an adult male Belgian hare, well fed and not very timid. While deeply under chloroform, an opening was made in the skull by means of a small trephine over the left prefrontal lobe, and through this the cerebro-spinal fluid was slowly aspirated, to prevent a too sudden withdrawal leading to rupture of the vessels, as occurred in Rabbit H. Only about two drams of fluid were obtained and this was slightly blood stained.

During the withdrawal the animal spasmodically jerked its limbs, in the same way as, but to a less extent than the animal last operated on.

While still under the influence of chloroform, a severe blow was dealt over the frontal region in a direction downwards and backwards, in a direct line towards the base of the skull.

The animal was thrown into a state of extreme opisthotonos with tonic contractions of all the limbs, lasting for a few seconds. This passed off, leaving only a distinct paralysis of the right forelimb. It was lying on its side. Some slight symptoms of compression gradually supervened but did not become serious. But for some drowsiness and deafness the animal seemed to recover completely, and was ultimately killed by chloroform inhalation.

Autopsy.—*Scalp.*—The incision in the scalp had healed by first intention. There was no ecchymosis into the scalp in the region of the blow. *Skull.*—The trephine opening was found to be over the left frontal lobe about the level of the anterior edge of the orbit. It was filled by a small mass of purulent looking material. No fracture of vault, and no contusion of pericranium. At the base there was a transverse fracture right through the body of the sphenoid bone, which permitted a bristle to be easily passed through into the pharynx.

Membranes.—Between the dura and the bone on the right side, there was a thin clot about the size of a split pea, situated on the upper aspect of the orbital plate. This clot was partly decolorised.

Hemispheres.—Just beneath the seat of the trephine opening, *i.e.*, over the left olfactory lobe, and inside the dura was a small clot, presumably due to the needle having injured the brain. The whole upper aspect was normal, and the lower paler than normal. No evidence whatever of blood clot either old or recent.

Cerebellum.—Normal.

Interior of Brain and Ventricles.—Normal. In the aqueduct of Sylvius was what appeared to be a small decolorised clot.

Bulb and Spinal Cord.—In the upper part of the spinal canal there had been some effusion outside the membranes, but it had been small in quantity, and by the time of death was nearly all absorbed.

Eyes.—There was no extravasation into the sheath of either optic nerve, and no subconjunctival ecchymosis.

Ears.—*Right*: Large red clot filling up middle ear. Membrane ruptured. *Left*: Partly decolorised clot in middle ear. Canals normal. Membrane ruptured.

Remarks.—On comparing this experiment with the last, we see that although in both the cerebro-spinal fluid was aspirated, the results differ considerably, and I attribute this to the varying rapidity with which the fluid was withdrawn. In rabbit H the fluid supporting the blood-vessels was rapidly withdrawn, while in this case it was slowly removed by gentle aspiration, thus giving the vessels time to adapt themselves so as to compensate for their loss of support. This, I take it, bears out the theory that in concussion the hæmorrhages found in such widely separated parts of the brain are due to this rupture of vessels suddenly deprived of the support of the cerebro-spinal fluid.

That the rôle of the cerebro-spinal fluid is an all-important one in concussion is further borne out by the result of the blow given to the head after removal of the fluid. If we look at cases of rabbits, pigs and birds, in every way identical, save in this one particular, that the fluid has been withdrawn, we find that while in them abundant lesions in the shape of hæmorrhages on the surface of the hemispheres, bulb and cord, into the substance of the parts, and into the ventricles occur, it is only very rarely that we have fracture of the strong base of the skull. Here, however, the conditions are entirely

reversed, because while we can only find two hæmorrhages on the surface attributable to the blow, and both of these external to the dura, and the ear lesions, there is a complete fracture of the basi-sphenoid. Let me point out that this process lies directly in the line of the axis of percussion, and would therefore be at the seat of the "cone of bulging." The cerebro-spinal fluid not being available to protect it, the force has spent itself on the bone.

In spite of the absence of the fluid however, there were some hæmorrhages resulting from the blow, *e.g.*, hæmorrhages into both middle ears, with none into internal auditory meatus. These can be explained on the theory that they were secondary to the fracture of the base. It is to be noted that the other extravasations are all outside the dura, and therefore more likely to be due to fracture than to cerebro-spinal disturbance. The bleeding from the mouth and nose also were doubtless secondary to the basal fracture.

The evidences of deafness exhibited all along, are of course quite intelligible in the light of the *post-mortem* appearances.

The absence of extravasation into the sheaths of the optic and auditory nerves, is due to the want of the cerebro-spinal fluid. At the moment of percussion these perineural spaces were not dilated, and so their vessels were not put into the dangerous condition which leads to rupture.

As a contrast experiment to the above, let us look at that performed on Rabbit C. In this experiment the blow was administered on the occipital region, but in every other respect, save that the cerebro-spinal fluid was not withdrawn, the two cases are similar.

Experiment 3, Rabbit C.—To compare results of severe blow to head with cerebro-spinal fluid in situ, with those of previous experiments where it had been aspirated.

November 30th, 1890, 10.45 a.m.—The subject was an adult grey rabbit, not in very good condition, but quite active.

Before any blow was administered the animal sat quietly, its pupils were equal, pulse small, rapid, 180; respiration also rapid, 90.

Anæsthesia having been induced by chloroform, a severe blow, intended to be lethal, was dealt over the middle line in the

occipital region, with the knob of a Kaffir stick. Immediately the creature fell on its right side, all the limbs were spasmodically convulsed for a few seconds. The animal then rolled over and over around its long axis five or six times, and came to rest on its left side. While in this position all the limbs were making violent movements similar in character to those made by an animal galloping, only very much exaggerated. For the first few seconds the fore paws were rigidly extended, but they soon took on the same actions as the hind ones. The head was dorsiflexed, and the tail extended in a straight line with the body. The pupils were equally contracted. This condition lasted for two minutes, then the limbs assumed different positions; front limbs were rigidly extended, and if flexed passively they immediately assumed their former position. The right hind limb was in the same attitude, the rigidity, however, being less marked, while the left hind leg was flexed; the head remained dorsiflexed; pulse full, bounding 120; respiration 124, shallow, very irregular and intermittent, mainly costal and noisy, whistling through the nares.

The pupils subsequently dilated equally, and the limbs exhibited regular and rythmical movements as of walking. The heart became weaker, and the respiration more difficult, and the animal was killed by chloroform inhalation.

Autopsy.—*Scalp*: Large hæmatoma over occiput and extending down as far as level of third cervical vertebra. *Skull*: Depressed and comminuted fracture of the right occipital bone; no basal fracture.

Membranes.—Only slight hæmorrhage from fracture between dura and bone.

Hemispheres.—Under the dura over the left cerebral lobe is a large extravasation, consisting of several large clots with thinner clots between; a smaller clot over the right lobe.

Cerebellum.—Over the middle lobe of the cerebellum is a blood clot of considerable thickness. It extends on to both lateral lobes, and is found to be continuous with an intraventricular extravasation described below.

Interior of Hemispheres and Ventricles.—The left lateral ventricle is filled completely by a large clot which is found to be continuous with that lying on the posterior aspect of the occipital lobe, and on the cerebellum.

This same clot extends into the third ventricle. A clot of considerable size fills the cavity of the fourth ventricle, and on being removed exposes several smaller extravasations into the

substance of the floor of that cavity. The part of the cerebellum forming the roof of the fourth ventricle is pulpy and infiltrated with blood.

The Sylvian aqueduct is enormously dilated, almost sufficient to admit a toothpick. It is filled with clot. There is a small hæmorrhage into the substance of the anterior corpus quadrigeminum of left side.

No hæmorrhage into basal ganglia.

Bulb and Cord.—Various hæmorrhages on base of brain. There is a tear in the substance of the medulla on right side, but no visible hæmorrhages into substance beyond those mentioned. Some extravasation into upper part of spinal cord.

Eyes.—Well marked extravasation of blood into optic nerve sheaths of both eyes, extending backwards from the eyeballs for about half an inch.

Ears.—*Right*: Some extravasation into the first part of the auditory nerve sheath, in the internal auditory meatus. Canals and internal ear normal. *Left*: Numerous well marked points of ecchymosis into the membranous semi-circular canals, and also hæmorrhages into the middle and internal ear.

REMARKS.—We may compare in a general way the two experiments on rabbits I and C. In the former the cerebro-spinal fluid was aspirated before the blow was dealt, in the latter it was left, and the result of that blow is of the greatest possible interest, because, while in rabbit I the brain tissue practically escaped serious harm, in rabbit C the gross lesions found in the substance of the brain were so numerous, and so widely spread as to be spoken of without exaggeration as universal. On the other hand, in the case where the fluid was absent the base of the skull was fractured, while in the other it was uninjured. When we consider that the intensity of the blow in these two cases was as near as may be equal, the results of the experiments lend great support to the cerebro-spinal fluid theory of cerebral trauma.

If we now compare the result of experiment on rabbit C with that on rabbit H, in which the cerebro-spinal fluid was rapidly withdrawn, we find that in the two the symptoms during life and the *post-mortem* appearances, so closely correspond, that we must consider that in both, the lesions are produced by the same mechanism, a mechanism differ-

ing in the two cases only in the means by which it is started. With so many gross lesions any one of which might produce symptoms, it is useless to attempt either to assign a symptom to each lesion or to each symptom a lesion. Rather must we look upon the injuries to the brain tissue as an index of the severity of the blow, and not as the definable causes of the phenomena.

The presence of blood extravasations into (*a*) the sheath of the optic nerves; (*b*) the sheath of the auditory nerves; (*c*) the aqueduct of Sylvius; (*d*) the lateral, third and fourth ventricles, is of importance for my argument as showing the rôle of the cerebro-spinal fluid in the production of lesions in cases of concussion.

I have thus shown by experiment that sudden aspiration of cerebro-spinal fluid leads to extravasation of blood; that slow aspiration does not do so; that a blow on the skull after the fluid has been so removed causes much less destructive lesions than when that fluid has been left in its normal position.

THE RÔLE OF CEREBRO-SPINAL FLUID IN PRODUCING HÆMORRHAGE INTO NERVE SHEATHS.

I have noticed in several cases that, as a result of severe blows, there were found extravasations of blood into the sheaths of different cranial nerves, and this fact I think throws considerable light upon the mechanism of head injuries. It is an anatomical fact of some importance that the cranial nerves have a reflection of the arachnoid membrane accompanying them as far as their foramina of exit from the skull.

Axel Key and Retzius (32) have shown by their injection experiments that the coloured liquid can be made to penetrate into the lymphatic channels which Ranvier (50) has shewn to exist around the fasciculi of nerves, and Schwalbe (33) has demonstrated that these intra-nervous channels communicate with serous or lymphatic spaces of the organs of sense. He states that, "these intra-nervous, serous canals communicate with certain spaces in the organs of

special sense, which we have described by the name of serous spaces or lymphatics. Besides, by the internal auditory canal the serous canals of the auditory nerve are in communication with the spaces filled with peri-lymph contained between the osseous and membranous labyrinth; by the optic foramen the serous spaces of the lamina fusca which accompany the vessels of the choroid, and communicate with the anterior chamber of the eye, are in relation with the serous canals of the optic nerve, and with the central lake at the level of the chiasma; an analogous arrangement exists in connection with the olfactory nerves."

In this relation also, Duret announces this general law, that "wherever we have a nervous element and a blood vessel nourishing it, around the vessel is disposed a serous space destined to receive in the case of excessive tension in the latter the aqueous part of the blood which it contains."

This being so, it is evident that the wave set up in the cerebro-spinal fluid by a blow impinging on the skull, will be transmitted along these peri-vascular channels, and cause distension of them, which will result in rupture and extravasation when the fluid recedes.

It appeared to me that if this fluid could escape from the nerve sheath, and thus obviate its distension, such hæmorrhages would not occur, and to test this point I performed experiment 4.

Experiment 4, Rabbit F.—To determine the rôle of cerebro-spinal fluid in producing hæmorrhage into nerve-sheaths, especially those of optic nerves; and other points.

Subject, an adult male Belgian hare in good condition. Both pupils equal and of medium size before experiment. The animal was put fully under the influence of chloroform, and then the left eye was enucleated, the optic nerve being cut as cleanly, and as far from the eyeball as possible with curved scissors. The orbit was washed clear of all blood, and hæmorrhage stopped by plugging. The animal, still deeply anæsthetised, a severe blow intended to be lethal was delivered on the occiput, having a direction from behind forwards, as nearly as possible in the axis of the optic nerves.

Death was almost instantaneous, no other movement than a

slight twitching of the right hind leg taking place after the blow. The right pupil was much contracted after the injury was inflicted.

Autopsy.—*Scalp*: Slight ecchymosis under the skin over the occipital bone in middle line. No wound of scalp. *Cranium*: No fracture of vault or base.

Membranes.—No hæmorrhage external to dura. Under the dura over the right cerebral lobe was an extensive but very thin clot. None on left side.

Hemispheres.—Extensive hæmorrhages into the lateral ventricles on both sides.

Cerebellum.—Much injured, covered with blood clot, and on right side more or less pulpy, blood clot and brain tissue being mixed up.

Bulb.—Laceration of wall, and extravasation of blood around the aqueduct of Sylvius, and into substance of corpora quadrigemina. Large blood clot on front of pons and medulla pressing on the superficial origins of the vagi, spinal accessory, and hypoglossal nerves. The floor of fourth ventricle exhibited numerous small petechial hæmorrhages, scattered over it in all its areas. The left restiform body was covered by a large thick blood clot; a smaller one existed further down on same side.

Cord.—Extensive hæmorrhage extended down the cord as far as the upper lumbar region, especially on left side, but also marked on right.

Eyes.—*Right*: Pupil normal after death. There was a considerable extravasation of blood into the back of right orbit, a clot about size of a threepenny piece pressing on the branches of third nerve. Sheath of optic nerve filled with blood clot. *Left*: The optic nerve had been severed about one-eighth of an inch from the eyeball, and there was no blood around the stump. There was no hæmorrhage along its sheath whatever.

Ears.—No hæmorrhage or escape of cerebro-spinal fluid from either ear. Both membranes intact. *Right*: Some blood in the sheath of the nerve. A few spots in the semicircular canals. None in cochlea or middle ear. *Left*: No hæmorrhage along course of auditory nerve in internal meatus. Semicircular canals filled by a large extravasation.

Jaws.—*Right*: At the inferior dental foramen by which the branch of the third division of the fifth nerve enters the bone was a fairly large clot of blood in the sheath of the nerve. *Left*: One small hæmorrhage close to foramen, and another, a short distance higher up the nerve.

Remarks.—There can be little doubt that the theory enunciated as to the origin of these hæmorrhages into the sheath of the optic nerve, was the correct one, because in the left eye the conditions were such that the cerebro-spinal fluid rushing along the sheath could escape without dilating it, and no hæmorrhage resulted; while on the right side the distension was followed by a copious hæmorrhage.

Not only in the sheaths of the optic nerves have we this recession of cerebro-spinal fluid, but also in the auditory nerves, fifth nerves, and probably in all the others the same thing happens, as is evidenced by the hæmorrhages found in these various positions.

Referring to the extravasations into the internal ears and along the course of the auditory nerves, one can easily understand how, if the tympanic membranes had been ruptured or previously perforated, a symptom supposed to be characteristic and diagnostic of fracture of the base of the skull, namely bleeding from the ears and “welling” of cerebro-spinal fluid, might have been present while no such fracture existed.

The lesions of the left side of the brain are much less marked than those of the right side, and it appears to me not improbable that this is due to a certain amount of the fluid finding a point of escape through the severed optic nerve sheath of that side, so diminishing the size of the wave set up by the blow.

SUMMARY.

Such are the grounds on which I am led to support and confirm this theory of the important part played by the cerebro-spinal fluid in the mechanism of cerebral concussion and other forms of cerebral trauma.

The conclusions I have formed are briefly these:—

(1) That the group of phenomena, commonly spoken of as “concussion of the brain,” is the result of a temporary anæmia of that organ.

(2) That this anæmia is the reflex result of stimulation of the restiform bodies, and perhaps other important centres in the region of the bulb.

(3) That these parts are stimulated by the wave of cerebro-spinal fluid, which rushes through the aqueduct of Sylvius, the foramen of Magendie, and from the subarachnoid space of the brain to that of the cord when a severe blow is dealt over the skull.

(4) That in accordance with the laws of hydrostatics this cerebro-spinal fluid wave will disturb the equilibrium of the ultimate nerve cells throughout the central nervous system.

(5) That the hæmorrhages found throughout the brain substance and on its surface are to be ascribed to the recession of the cerebro-spinal fluid, which naturally supports the blood vessels of the cerebrum.

(6) That the petechial hæmorrhages found in cases of so-called concussion are not the proximate cause of the symptoms of that condition. They are rather to be looked upon as an index of the force producing the injury, than as the cause of the resulting phenomena.

In another paper (48) I have described the microscopic appearances found in these and other cases of cerebral trauma, most of which, I feel sure, may be satisfactorily explained on the same theory as are the grosser lesions to which I have alone referred in the foregoing paper.

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THE ROLANDIC AREA CORTEX.¹

BY EUGENE DUPUY, M.D. (PARIS).

SINCE the epoch-making "Researches into the Physiology of the Brain" of Hitzig and Fritsch, and the later labours of Ferrier and the numerous physiologists who have followed, it has been accepted as firmly established that the grey matter of the brain—the cortex—is not only excitable by electricity, but that well-defined centres of motor action are contained in certain definite areas of the convolutions chiefly around the Rolandic fissure, or its analogue in the brains of lower animals. I have from time to time since 1873, endeavoured to show that the proofs brought forward were not sufficient; and that the facts observed by the different experimenters could be explained more satisfactorily as purely physical not physiological, phenomena.

(1) It has not been demonstrated that any other agent but electricity does produce any effect in the motor apparatus of animals when applied to the cortex; and this agent cannot have a well limited field of action, since it diffuses through lines of least resistance. Physical, chemical, or mechanical agents fail entirely to give rise to motor action when used in the same manner as electricity, while it is well known that these last agents all affect nerve fibres. The fact that when a current of minimum strength applied to certain spots in the cortex of the brain of certain animals (species) the experimenter is able to predict the effect to follow, only goes to show that certain points of the grey matter of the convolutions are situated in the line of the least resis-

¹ Paper read before the Neurological Society of London.

tance. In reality, all brains of the same species are furrowed by sulci and offer the same mode of arrangement; as these contain elements which constitute good channels of conduction, it happens that on brains in which such similar circumstances obtain, identical results must follow. Moreover, the pia mater over the Rolandic convolutions is almost a complete network¹ of vaso-motor fibres and cells, and blood-vessels, which penetrate into the convolutions. One system of blood-vessels only ramifies and ends in the cortex proper, the other enters the white substance by means of larger vessels accompanied by nerves and ganglion cells. It follows from this arrangement that the cortex proper is much more vascular and the white substance or fibres a great deal less so; and moreover, that whilst the vessels irrigating the cortex are spread well over, those going into the white substance are isolated from one another. Now I have shown long ago² that those points, which when "excited" by electricity give rise to a motor action, coincide with spots where arteries with nerves penetrate into the white matter or strands of fibres. The best method to demonstrate this fact consists in injecting one carotid artery after having marked with ink the position of the "motor centres," ascertained beforehand by means of electricity.

Experiments, well known to all, are appealed to not only as giving a complete demonstration that the cortex is excitable (around the fissure of Rolando), but also as proving the impossibility of the explanation I have given. Putnam made this one experiment: after having found the minimum current sufficient to excite motor action when applied to a certain group of "motor centres," he slices off the cortex, leaving it *in situ*, and then, on repeating his excitation, he finds that the same current is insufficient to call forth motor action. Now this experiment is anything but conclusive, vary it as much as one wishes. First, the physical conditions of the two experiments being different, we can by no means obtain identical results;

¹ Hénocque *Thèse inaugurale. Des terminaisons nerveuses.* Duret, *Circulation de l'Encéphale*, 1874.

² Dupuy. *C. R. Soc. de Biologie*, 1887.

indeed, the slicing off of the cortex and the consequent clotting of blood, and the creation of two moist surfaces, evidently alter the distribution of the current; it spreads more, and must therefore be strengthened to give the same results as before. Secondly, I have shown that if the cortex being sliced off and the same minimum current used to ascertain the production of motor action be applied not directly, but after a short time, to the cut surface of the fibres, results are obtained identical with those in the first experiments, when using the same current as applied to the cortex proper before the slicing off; this indeed was to be expected, it proves that fibres below the cortex are excitable, it neither proves nor disproves an excitability of grey matter proper.

Another experiment devised by Frank and Pitres consists in marking the time elapsing between the moment of application of a minimum current to a "motor centre" and the occurrence of motor action; and they found that there is a difference in *plus* equivalent to about one six-hundredth of a second upon the same time elapsing from the moment of application of the same current to the cut surface of white matter (the cortex of which has been sliced off) and motor action following. This longer time they ascribe to the elaboration of nerve force by the cells or reaction time. Bubnoff and Heidenhain and others have confirmed these results. The two last named observers have moreover discovered this remarkable fact, that slight irritation of the skin of the limb of which the "motor centre" is being experimented upon, increases the excitability of this centre when the animal is under the effects of anæsthesia and morphinisation; but from what I have already said, it appears clear that the reaction time of Frank and Pitres is owing entirely to diffusion of electricity in a tissue much more vascular than the white substance fibres under the convolution, as the cortex proper is known to be; and that there takes place a series of actions almost inevitable (electrolysis, short circuiting, &c.), hence the difference in the results. As for the explanation of the fact discovered by Bubnoff and Heidenhain, I find that when they excite

the sensitive nerves of a limb (skin) they certainly bring on vaso-motor change in the brain surface, and since anæsthesia and morphinisation induce contraction of blood-vessels, therefore less nutrition and excitability of nerve tissue, the irritation of the skin of the limb changes that state by *plus*, as I have ascertained in the pupil of the eye which shows the characteristic reaction, known since the early days of Vulpian and Schiff. The facts recorded so far appear to me to allow more than strong doubts being entertained as to the excitability of the cortex proper by electricity, it being inexcitable by any other means. The experiments devised by Frank¹ and consisting in circumvallation of a given centre, go a long way to support my theory. Frank says that: "Having laid bare the right hemisphere of a dog, it is noticed that the animal, after the sigmoid gyrus has been uncovered by section of the dura, behaves exactly like a well animal; the right hind leg appears a little more outward than its fellow. The animal attempts to get rid of the muzzle by means of its left fore paw chiefly. The motor centre for this limb having been ascertained by electricity, a bistouri is passed around the motor area to the white substance, describing a circle around the gyrus, the depth of the incision notably exceeding the depth of the grey matter; *care has been taken to save two bridges of pia mater containing large arterial and veinous vessels.*"² The animal left free behaves as before and uses his two fore limbs to get rid of the muzzle, *but just as before the "circumvallation" of the motor centre of this limb, it is the left fore paw which he uses chiefly.* A weak stimulation of that motor centre which is now isolated from the rest of the cortex by the circular incision, at first gives rise to localised motor action in the left fore paw, and then an attack of convulsions which become general, invading all the limbs and lasting a long time, etc.

After renewed weak electric excitations, which show the persistency of the excitability of the circumscribed area of

¹ Frank. "Fonctions motrices du cerveau," p. 459. Marique. *Thèse*.

² No italics in text.

cortex, the whole of the grey matter of this area is sliced off, the white surface thus produced being dried up, it is found that it requires a much stronger current to induce feeble motor action in the left anterior paw; the animal falls over the left side and does not succeed in getting upon its legs when left on the floor. In a second animal the same experiment is performed, but in order to avoid flow of blood which had proved embarrassing, the circumvallation of "motor centre" was made with a galvanic cautery (the centre for the left anterior leg) and in such a manner as *to save the large veins*; the section is completed with a bistouri. When left on the floor the animal shows manifest motor troubles on the left side, chiefly the fore left leg. Electrical irritation of that area thus isolated fails to give rise to motor action even with currents progressively strengthened. After removal of the cortex it is found that the white matter is only excitable much deeper, &c. A third experiment is made upon another animal, and the whole sigmoid gyrus on the right side is circumvallated by a bistouri section, but continuity of the superior portion with the rest of the brain is preserved. No trouble of motion whatsoever is observed. The section is completed so as to form an island of the gyrus, and the animal now shows manifest signs of motor troubles in the left limbs, &c., and excitation of the circumscribed area brings forth evident motor action in the left anterior leg (crossed action). One hour later, the motor troubles had greatly diminished, the gait being less awkward, &c.

It appears to me, from these results, that the theory I have advanced (the action of electricity upon white matter through blood-vessels and nerves accompanying) is well supported, and the cause why in one experiment (No. 2) it was necessary to irritate a deeper layer of white matter is only due to the fact that the upper layers of white substance receive their blood-vessels from the cortex, and the lower by the opto-striated system of vessels; and the galvano-cautery action was more than enough to account for the shock effect produced on the white substance, besides coagulation inevitable when cautery is used. I must here state that in

dogs at least the blood supply of the convolutions appear to be less differentiated than in man, for it is impossible not to fill all the arteries of one hemisphere (convolutional chiefly), when colouring matter is pushed through either carotid, all the arteries seem to communicate in the cortex. Frank has criticised my views on the rôle played by penetrating arteries and nerves in the effect of exciting the strands of white fibres when electricity is applied to "motor centres." If he had endeavoured to understand my view he would have seen that my meaning is simply that these elements offer lines of least resistance; and now that we possess the results of the researches of Golgi and his school, it is quite easy to understand that if the blood-vessels with which the protoplasmic prolongation of cells are in contact is undergoing alteration, the nerve elements will also undergo change. And in last resort we have to deal here with a simple case of animal electricity, positive or negative variation as the case may be, and induced by change in blood supply. When Kussmaul and Tenner, having tied the four blood-vessels going to brain, find that convulsions supervene, they have produced exactly the same change which I claim we produce when we use electricity on 'motor centres,' *i.e.*, electrical variation. I simply stated that the current (minimum) only acted on the "motor point," which is also the point of penetration of the arteries accompanied by nerves; I never stated that the current went all along the vascular supply and selected one artery and its nerves, and therefore I see no reason to ascribe to me the inane theory which Frank has criticised. I have preferred to give as illustration of my views by means of experiments performed by Frank himself, although it is needless to say, he did not insert the record of these for my purpose.

Another experiment of Frank's consists in freezing the "motor centre" and then irritating the hard frozen surface; it is found that whereas before the freezing the motor action in the limb was longer in duration than the current used and was epileptic, and besides only occurred after a lapse of about $6\frac{1}{2}$ -hundredths of a second, when the same current is applied during the frozen state the time of reaction is only

about $4\frac{1}{2}$ -hundredths of a second, and the contraction is only tonic. The claim is made from these results that the reaction time is greater ($6\frac{1}{2}$ -hundredths of a second) when the cortex is not frozen because it reacts *physiologically*, and the time is shorter ($4\frac{1}{2}$ -hundredths of a second) when the cortex is frozen because it reacts *physically*.

This I consider a very unacceptable interpretation; it is unnecessary to state that the white substance as well as the cortex is influenced by the freezing process and the irritability of both endangered and altered; besides, what of the inhibitory action of such a frozen "wedge" into the centrum ovale?

But I have shown that if two fine needles, insulated by varnish altogether and the points only shining, are used instead of ordinary electrodes, and these fixed in a cork and protruding one millimetre and a-half apart, and passed with care below the pia mater (in same manner that hypodermic needles are made to travel into the skin) the reaction time is identical in both cases, whether the electrical minimum currents be passed between the cortex and the pia or just below the cortex.

The figures given by Frank in his book¹ do not support his own theory. In the first graphic the tracing shows that after cortical freezing the contraction graphic line does not last much beyond the time of irritation, and yet the character of the contraction is epileptic. If the cortex reacted physically when frozen the contraction ought to have been purely tonic and to have lasted only as long as the current used, and not to have shown the character of tonic and clonic convulsion. Later, while the cortex was getting unfrozen, and still later when it was unfrozen, the tracings show an increase only in the duration of the epileptic contractions, a result which it is natural to expect, considering the state of change induced in the 'motor centre' area by the preceding freezing—it is inflamed. I much prefer to make use of facts recorded by those authors who hold the doctrines which I consider erroneous to show

¹ Frank. "Fonctions Motrices du cerveau," p. 113. The three graphics are very interesting.

how misinterpretation has led them to frame the doctrine of localisation of motor functions in the cortex. I call such facts "impartial."

Thus the statement that muscle contraction differs when the cortex is excited by being of longer duration than when the white fibres alone are excited, is proved to be erroneous by Frank himself in the experiments which I have reported above, and the value of the distinction in the nature of the muscle contraction, if it did really exist, would be negatived by the fact that complete epileptic convulsions occur even after the destruction of the cortex over the motor area as well as over the whole brain.¹ So far from denying I was the first to assert that soon after slicing off the cortex, electrical irritation of the artificial surface of white substance may give rise to a motor action identical with the one following electrical excitation of the cortex; indeed, nutrition of the elements is highly impaired and altered, and we must take into account the inhibitory action also arising from traumatism, but it remains constant that in this condition the limb, the so-called 'motor centre' of which has been sliced off, becomes the seat of epileptoid contractions, tonic and clonic. Ferrier² says that I have degraded the cells of the cortex of the 'motor centres' by stating that they are not the elements upon which electrical currents take effect. I deplore that I am guilty of that great offence; I shall however try to atone in some manner later. I do not believe that anybody can show that grey matter in the spinal cord can be excited into activity by any agent applied directly to it (not through nerves), and the grey matter of the cortex being not of a different nature from that of the spinal cord, there is no *a priori* reason to

¹ After having ascertained 'the reaction time' when the cortex is irritated by electricity, and also when the white substance uncovered by slicing off the cortex, a thin piece of amadou is placed over the white substance in place of the cortex—amadou soaked with cerebro-spinal fluid or physiological salt solution, and the 'reaction time' when electricity is applied to this artificial cortex is apparently the same as when electricity is applied to the natural cortex. It would be rather absurd to speak of amadou reaction time I imagine, although in the case of amadou, as in the case of the cortex, the reaction time is apparently the same, and the muscular response is alike in both instances, being epileptic (tonic and clonic).

² Ferrier. "Localisation of Cerebral Disease," p. 16.

suppose that it should be endowed with different properties. The cells of the cortex may well remain undegraded, even if we consider them as Golgi has been teaching, and with him all his school and chiefly Nansen,¹ to be trophic elements ; or if we attribute to them different functions than those ascribed to them by Ferrier. There are besides, as I will show in a moment, so many reasons to reject the theory that the cells are either motor or sensory, and that there are in certain areas of the cortex 'motor' or 'sensory' centres, that this doctrine is no more tenable. Already great differences exist between those who teach the doctrine of 'localisation of functions' on the interpretation of the nature of the very function of the cortex. It is not necessary to mention before this Association the hypotheses of Hitzig, and of Nothnagel, who consider the Rolandic region as the centre of muscular sense or muscular consciousness ; of Bastian, who separates muscular sense from the other compounds which go to form his 'kinæsthesia' ; of Schiff, who believes that it is some kind of sensitive region in connection with tactile sensation, said by him to travel through the posterior spinal columns ; nor the doctrine of Ferrier, who considers the Rolandic region as entirely motor—a doctrine which is identical with the one taught by Hughlings Jackson (the original founder of the doctrine) ; nor of Charcot, who holds that the "motor centres" are the substrata of motor action as distinguished from sensation and sensory centres ; nor Munk's psychic theory of sensory spheres representing images of the different species of sensations common and special, and which react in a psychic manner—"metaphysiological," if I may so express it. There are also a number of theories, chiefly evolved by the Italians, which are modifications of those given above. And I have only thus briefly enumerated them to give prominence to the want of agreement as to the specific nature of the 'functions' of the cortex of the Rolandic region.

But a more practical question is this one : do the nerves which set into action the limbs of the animal experimented

¹ Nansen. *Nordiskt. Medicinskt. Archiv.*, 1887, xix. 4, p. 1-24.

upon possess a centre of whichever nature 'sensori-motor,' or 'kinæsthetic,' or purely 'motor,' &c.—always well defined, occupying in every brain of the same species of animal, the same area of the Rolandic convolutions? To this question I can give a more qualified answer, and will do so by means of a few chosen, and I consider, crucial experiments as well as by means of some facts gathered from human pathology.

Having sliced off the 'motor' area in the brain of a rabbit¹ I allowed it to rest for some time, and then performing Kussmaul and Turner's experiment of ligaturing carotid and vertebral vessels, I find that epileptic convulsions supervene at the same time in all four limbs.

In another experiment, also on a rabbit, only one motor area is destroyed, and it is found that the results of Kussmaul and Tenner's experiment are also identical in all four limbs.

In a dog² I performed the operation of removal of the sigmoid gyri on both hemispheres in Feb., 1886, and in April of the same year it was examined by a committee of the Biological Society and the report drawn up by Prof. Mathias Duval. I extract: "The gait appears very slightly choreic, that is to say that the animal walks balancing on either side, rather undulating like the tail of a fish; the hind limbs are slightly apart—in fact, very little disturbance, since the anterior limbs are perfectly normal. The left eye, which was the more troubled, permits good vision enough to avoid some obstacles.³ Placed upon a table the animal jumps on the floor after the manner of a dog in the natural condition. As the examination fatigues him (by their duration) and worries him, he walks a little less well, slipping slightly upon the plank floor, the limbs apart slightly. Hearing is good. A lump of sugar being thrown at him he picks it up and searches for scattered particles on the floor. Examination with Dubois-Reymond's coil gives, when the coil is 10c. removed from

¹ Dupuy. "Physiology of the Brain," &c. *New York Medical Journal*, May, 1877.

² Mathias Duval, Rapport, &c. *C. R. Soc. de Biol.*, p. 371, 1886.

³ There are always visual troubles in experiments on the cortex.

the core, good sensation in the right limb; at 30c. the same limb feels still but slightly; on the left limb he feels even a little with a removal of the coil to 40c. The hind limb (left) feels very well with coil at 30c., the right is still more sensitive with the coil at 30c. Electric irritation is therefore better felt on the left fore limb and right hind limb, as compared with the right fore and left hind limbs. By pressure with forceps sensation appears to be a little less on the right than on the left limb. The fore limbs feel equally well. That dog appears to be rather sensitive, or simply perhaps a little 'douillet.' It is killed by section of the abdominal aorta. . . . The brain, on examination, is shown to be adherent to the skin healed over the original wound, and by means of cicatricial tissue. On the right hemisphere there is a scar covering the three-fourths external posterior of the sigmoid gyrus; that scar covers also the convolutions immediately in front and behind. On the left hemisphere the scar is alike, only a little more extended in the antero-posterior area. . . . At the level of the scars the cerebral matter is rather much depressed, the border drawn towards the centre. After maceration in alcohol sections are made, which show on the right hemisphere ('il saute aux yeux') that the sigmoid gyrus has been completely removed, in the place of its deep portion (sulcus) cicatricial tissue is discovered which invades into the white central matter. On the left hemisphere the sigmoid gyrus grey substance appears also to have been completely removed, but the subjacent cicatricial tissue extends much less in depth, and part of the white matter of the gyrus appears to have been spared. Microscopic examination shows the fibroid cicatricial tissue infiltrated by granular elements. As said above, it is seen also that the scar on the left gyrus was more extended antero-posteriorly and, in fact, the sections show that the grey substance has been removed from the frontal convolution immediately in front of the gyrus."

Another dog was subsequently operated upon by me and shown to the Biological Society at several later meetings, and the results were in every manner similar. The brain was examined by Mr. Vignal. I have not alluded chiefly to the

experiments and results of Goltz because the reports of these published by the English Committee, as well as other reports published at Berlin later, show that although he has removed large areas of cortex, he has nevertheless, spared portions of the sigmoid gyrus cortex in one or in both hemispheres. But the brain examined by the Biological Society Committee, as said above, showed that the two gyri have been completely removed as I announced. In human pathology, since the new era of operating upon 'motor centres' has begun in earnest, literature records very interesting observations—I find a number of illustrations of facts identical with those obtained by my experiment on the dog. The following are taken of a greater number collected from journals, periodicals and memoirs easily obtainable:—

(1) Epilepsy induced by focal lesion in facio-lingual "motor centre." Operation performed. Removal of cyst from lower part of ascending frontal, size of a filbert, and situated partly in cortical and partly in white substances of the brain, and surrounded by a narrow zone of encephalitis. The muscles were paralysed after the fits (tongue, right facial, and platysma muscle). Complete recovery after operation.¹

(2) Left-sided paralysis, arm and leg. Operation. Removal of gummous fluid with pultaceous particles and osteoplates. Later, second operation. Power of arm and leg restored so as to walk, perform household duties.²

(3) Paralysis of left hand and wrist, and epileptic. Operation upon cortex at level of fissure of Rolando but posteriorly, a piece of bone is removed which compressed the brain, which was found altered. A cyst is removed, and also about two teaspoonfuls of cerebral substance. After five days the patient was in good state. Paralysis relieved.³

(4) Convulsions, later hemiplegia, right side. Half an ounce of pus is removed from the ascending frontal incised one inch deep. Motor power recovered in arm and leg.⁴

¹ McEwen. *Lancet*, p. 257-8, 1888, with figure.

² *Id.*

³ Keen. *Internat. Journal of Med. Science*, Nov. 4, 1888.

⁴ *Liverpool Medico-Chirurgical Journal*, p. 303 1888.

(5) Epilepsy and left hemiplegia. Middle portion of fissure of Rolando is laid bare, and meninges and cortex diseased are removed. Complete recovery. Recovery of motor power.¹

(6) Paresis of right leg, contracture of right hand, and epilepsy. Operation. Excision of lining of old lesion occupying substance of the ascending frontal. The excision was wide. Relieved from contracture and recovery, in a great measure of paresis.²

(7) Epilepsy. Right arm enfeebled after attack. Operation. 'Motor centre' circular incision made five millimetres around a cavity found in cerebral substance, and which is five centimetres deep and one centimetre wide. Paralysis disappeared three months later.³

(8) Epilepsy, right hemiplegia. Operation on superior third of ascending frontal. Very vascular scar three by two centimetres. It is excised together with five millimetres of surrounding substance and a little over two centimetres in depth. Relief from paralysis.⁴

Although I am dealing only with "motor centre doctrines" as "illustrated" by experiments made upon animals and surgical operations upon men, I will give some cases of disease of the "motor region" in man, since it is claimed by the anatomo-pathological school that nature performs far more precious experiments than experimenters can in their laboratories. The following are quoted from a number:—

(1) Atrophy of the upper thirds of left ascending convolutions. No paralysis right or left limbs.⁵

(2) Sarcoma growing from dura having apparently destroyed the greater part of motor area on right side, so far as one could judge with naked eye, the whole of motor centres for face and upper limb are destroyed. Microscopical examination shows that grey substance of this region seems

¹ *Congres français Chirurgie*, p. 308, 1885-6.

² *Bulletin de l'Academie de Medecin*, 1889, Aout. Lucas Championnière.

³ Horsley. *British Med. Journal*, p. 674, 1886.

⁴ *Id.* p. 672.

⁵ Magnan. *C. R. Societ. de Biologie : Memoirs*, vol. xxx., p. 73, 1878.

to have completely disappeared, and yet there was absolutely no paralysis.¹

As a number of cases, great indeed, can be found in literature, I will not quote more here, and these I have taken from Anglo-American literature mostly, as I am addressing an English Association.

Although aphasia is not my subject, yet since Broca's convolution is considered to be the seat of motor aphasia *par excellence*, and in the absence of which no speech can take place, and whereas it is taught that word-blindness, word-deafness, may exist with conservation of speech, if the occipital or temporo-sphenoidal lobes alone are altered, yet speech expression only persists so long as Broca's region is not destroyed.

(1) Patient was stricken down with right hemiplegia and aphasia eight years previously. She began to speak again at once, and in some manner suddenly, without having undergone preparatory re-education. About eighteen months after the attack of hemiplegia she could speak in a very precise manner—intellect intact. Autopsy. The left lobe is softened partly in the vascularisation area of the sylvian artery. The posterior portion of the third frontal (left) has been destroyed by softening, as also the insula, the junction of ascending frontal and parietal convolutions, and the first parietal are gone. In a word, the whole of the cortex of the lobe which make up the insula and marginal convolutions around it is destroyed. Other parts of the lobe are normal except grey substance of the corpus striatum, extra ventricular nucleus, which was destroyed. Intra-ventricular nucleus sound, marked atrophy of thalamus.²

(2) Paralysis of right upper and lower limbs, articulation thick and indistinct, patient complained of pain in paralysed limbs, emotional, noisy at night, &c. Left hemisphere sectioned, corpus striatum found softened to diffuence, also contiguous part of thalamus in limited area; softening involving from corpus striatum outward and upwards to surface including posterior part of third left frontal convolution.³

¹ Byrom Bramwell. *Brit. Med. Journ.*, April 21, 1898. Plate.

² Luys, in *C. R. Soc. de Biologie*, p. 330, vol. 28, 1876.

³ Allan. *Lancet*, May 2nd, 1885, p. 797.

(3) Patient (cardiac dropsy) not the least symptom of any defect of speech, nothing pointing to cerebral mischief, when spoken to would give correct and sensible replies. Autopsy. Atheromatous arteries. Third left frontal convolution almost entirely destroyed, along with two-thirds of island of Reil. Over this area pia mater disorganised and converted into non-vascular transparent shreddy membrane, adherent to pulpy-brown surface of irregular excavation where brain substance was missing, brain otherwise apparently free from disease.¹

(4) Patient suffering for three years from spasms of right side of face and later right upper limb, also lower limb; epileptic attacks. Answered questions rationally, though speech was thick. Autopsy. Medullary substance of left hemisphere reddened, line of cortex very indistinct, brain almost diffuent. A portion of this hemisphere (left), three inches long and one and a quarter deep, was thus affected. The ascending frontal and ascending parietal convolutions were implicated, the disease extending inward to the lateral ventricle; microscopic examination showed that glioma was the disease.²

There are a number of other cases of destruction of Broca's centre without aphasia (in right-handed persons) and even in Nothnagel's treatise on Cerebral Localisation (French translation) very fine examples are to be found, but he explains them by saying that aphasia did exist and was recovered from. That would according to him, confirm the rule.³ It all depends upon the manner of viewing the case, as I shall show later on.

It is, unfortunately, impossible to study aphasia by means of experiments upon animals, but it is fortunately possible to do so by means of surgical operations performed

¹ Dr. Foulis. *Brit. Med. Journ.*, March 15th, 1879, p. 383.

² Cheesman. "Archives of Medicine," p. 84, t. 2, vol. 6. Plate. I quote this case, although the author makes no mention of aphasia lesion of the third frontal, because the context shows that destruction of Broca's centre existed, as also demonstrated by the delineation of the lesion in the plate. It is an "impartial" case published for a purpose not connected with aphasia.

³ Nothnagel. "Traité clinique du diagnostic des Maladies des encéphale," p. 393.

upon man if I may trust to the accuracy of the following report :—

Three patients, under the charge of Burckardt were suffering from brain disease with that form of delirium present, which ends ordinarily in dementia. B. trephined with the idea of stopping the hallucinations by suppressing the speech centre in Broca's convolution. The desired result was obtained and sensory aphasia, consequent upon the operation, disappeared rapidly in all cases. One patient became more quiet and able to work without being dement. A second patient was also improved, the third remained partially aphasic, and after leaving the asylum committed suicide.¹ I am aware, of course, that those cases of (motor) aphasia I have quoted are considered to prove not only that Broca's speech centre is the "one," but also that on it becoming impaired, the other, in the opposite hemisphere, comes into activity. Some experiments I will report further on do not afford much foundation for that theory.

Another question I must speak of is the one concerning the absolute necessity of the cortex to produce true epileptic convulsions—it is a corollary of the question of "motor centre" naturally.

I have already alluded to some experiments which I performed long ago, to show that the limbs of an animal can become convulsed at the same time and in the same manner as when the cortex is present even after removal, partial or total, of the 'cortex motor' area.

(1) Having laid bare the brain of a dog, I proceed to induce epilepsy by means of electrical irritation of one 'motor area,' and later on I slice off the cortex from 'motor area' of both hemispheres. Electric irritation produces, when applied upon the surface of white substance thus laid bare, epileptic convulsions identical with those observed in the first experiment.

After allowing the animal some time to rest, a further slicing off of the brain (both sides) is made, so that nothing remains in the cranial cavity but the large ganglia attached

¹ *Revue des Sciences Medicales* (Hayem's) t. 37, p. 592.

still to the pons. This last organ being irritated with electricity, epileptic convulsions supervene; very violent, and, in one instance, return without electrical irritation.

(2) In several animals (dogs), having sliced off the motor areas in both hemispheres, essence of absinthe is injected (into saphena vein); a most violent epileptic fit breaks out and the animal continues for four minutes to have fits with short intermissions. The brain is sliced off completely and the pons irritated as before, and epileptic fits again occur.

These results were to be expected from previous experience. It is many years now since Brown-Séquard has shown that even after removing the brain of guinea-pigs, which he had rendered epileptic by lesions of the cord or of the sciatic nerve, fits did and would occur. I have made the same experiments a number of times already long ago, and it is therefore to be expected by me *a fortiori*, that true epileptic attacks will come on in animals, even in the absence of the cortex of motor centre area, since in some experiments the brain proper having been cut off attacks broke out.

Human pathology offers similar observations, since there are many records of epileptic fits beginning in the limbs, the 'motor centres' of which having been found destroyed by disease. Seppili¹ observed a case of which the record is: "Patient 30 years old, epileptic for seventeen years; left hemiatrophy of arm which is completely paralysed; leg moves slightly, both contracted. The attacks begin by a feeling of formication in the fingers of left hand, at the same time almost clonic convulsion of the arm muscles occur, then the left leg is involved, and face (left side) without loss of consciousness; later, the other side of the body becomes involved. Sometimes the fit begins at the same time in the two sides of the body, and then there is loss of consciousness. After the fit the left limbs remain completely flaccid. Autopsy. Left hemisphere sound, right hemisphere smaller, membranes adherent over large area of the right hemisphere comprising the posterior portion of the three frontal and

¹ In Soury. "Fonctions du Cerveau," p. 421.

the two ascending convolutions, the paracentral lobule and the parietal convolutions. The lesions extended into white substance and reached the superior and external parietes of the right lateral ventricle. Consistency of brain substance very feeble in this zone. The cortex as well as medullary tracts were transformed into fibrous tissue formed by numerous partitions which, by intersecting, formed small cavities containing semi-gelatinous substance. Microscopic examination showed that no nervous elements existed in that zone, but a considerable amount of amyloid corpuscles and pigmented and fatty granulations. The membrane which gave to this region the aspect of areolar tissue was formed by connective elements; numerous sclerosed vessels existed in it, but the lumen obstructed by accumulated pigments. Moreover, there was degeneration of right pyramidal tract, traced into right internal capsule, cerebral peduncle, bulbar pyramid and left lateral column of cord; opto-striated bodies normal."

Seppili being a distinguished physician and one of those who hold the 'Doctrine of Localisation' to be so well founded, I consider this case very instructive.

Is it possible to explain away the cases of cortex lesion in the 'motor area,' say for one limb without paralysis existing on the hypothesis of vicarious function of the neighbourhood or of the cortical area of the same 'centre' in the other hemisphere? Although Brown-Séquard has shown by experiment that the divided fibres of corpus callosum are excitable, giving rise to motor action in one or the other hemisphere according to the surface of the section irritated; still the balance of evidence is against the hypothesis that 'suppleance' or vicarious action takes place in the 'motor centre' of the other hemisphere. Carville and Duret¹ have found that, if some time after destruction of 'motor centres' for the limbs of one side of an animal has been made and the animal has recovered from the awkwardness or paresis, the 'motor centres' for the same limbs of the other side of the body, and situated in the other hemisphere, be also destroyed, instead of a paralysis following in

¹ Carville and Duret. *Archives de Physiologie*, 1875, p.

the limbs at first the seat of motor alteration and which had recovered, the new operation induces motor trouble in the limbs which were not affected by the first lesion. I have showed already that destruction of "motor centres" in both hemispheres is not followed by paralysis in either side of the body. And moreover, if after the animal having lost "motor centres" say the sigmoid gyrus in one side and having recovered and using all four limbs equally well, the brain is again laid bare and electrical irritation applied to the cortex all around the scar and in healthy cortex therefore, no motor effect is observed to follow in that limb—I have shown¹ also that if electricity be applied to the white substance surface produced by the destruction of the cortex several days after the healing process is complete, no motor effect is obtained and yet in those instances of absence of cortex no paralysis is observed.

It is evident from these that the cortex has no vicarious activity, that the "centres" do not replace one another in the same hemisphere, or in the other hemisphere—a conclusion at which I am not at all astonished, as I do not consider that the cortex contains "centres" which govern motion at all in the sense admitted by most. It does not seem possible to admit that motor impulses are generated in the so-called "motor cells" of the cortex and travel necessarily through and into the pyramidal tracts and cross into the cord. The reasons are the following, as found by experiment :

I divide one cerebral peduncle in a dog, and after allowing the animal to rest, the "motor area" situated in the hemisphere of the same side is irritated with electricity. A complete epileptic fit occurs, beginning in the side of the body opposite to peduncle divided.² Brown Séquard³ had already found that after section of one cerebral peduncle or one half of the pons, electrical irritation of the "motor centre" of the same side was followed by motor action on

¹ Dupuy. *C. R. Soc. de Biol.*, 1875, in *Gazette Medicale de Paris*, 1875, p. 376.

² Dupuy. *C. R. Soc. de Biol.* for 1886, p. 19.

³ Brown-Séquard. Specially reported in *Archives de Physiologie*, 1889, p. 220.

the opposite side of the body. It is also demonstrated experimentally that after hemisection of the cord and recovery from the effects of trauma and paralysis, animals recover motor power in the parts which had been incapacitated, and I have found that if hemisection on the two halves of the cord (alternate sections) be made below the bulb at some little distance from each other, then electrical excitation of left or right cortical "motor area" or sigmoid gyrus of the dog, motor action takes place in the limbs below the sections.

It remains now to report another series of experiments. I have found that it is possible to induce epileptic convulsions in a dog even after electrical irritation of the brain fails to do so; as for instance when in the etherized dog (after it has been subjected to cortical irritation by electricity), morphine is injected so as to induce more complete anæsthesia, then the current applied to the cortical "motor area" fails to induce any motor action; the same irritation however applied to the dura is followed by an epileptic attack.¹

From the facts and arguments I have so far considered it does appear to me that the cortex of the "motor centres" are not necessarily the "centres" from which motor impulses are sent to the limbs, and before closing this line of argument I will report one last experiment.

Having laid bare the brain of a monkey under ether and morphine, the dura is split over one lobe so as to expose the "motor centre" for the opposite fore limb, and electrical irritation of that "centre" induces a fit of genuine Jacksonian epilepsy, and again, a second application induces a second attack, both having started and being localised in the same side, *i.e.*, opposite to the 'centre' irritated, but during the second attack the animal has recovered enough from the effects of narcotisation, and with the limbs not affected he grasps the sleeves of my coat so that by one half of his body he is holding on while the other is still epileptic. At this moment the fold of the dura which had been split and turned

¹ Dupuy. *C. R. Soc. de Biologie*, p. 339, vol. 41, 1889.

over to expose the 'motor centre' is irritated with the same electric current, and another fit of Jacksonian epilepsy follows, but this time in the limbs, with which the monkey was holding on. He looses his grasp, and while this second fit is going on the first is already recovered from.¹ Similar experiments made on a dog and on a cat gave the same results, and also the fits in some instances have become general after irritation of the dura, the cortex being inactive, a not unfrequent occurrence in Jacksonian epilepsy, the case quoted above from Seppili being an illustration in point, the patient had fits which were sometimes unilateral and sometimes general. It cannot be held, it appears to me, that the cells of the cortex of the brain constitute "motor centres." The experiments I have reported and the pathological facts do not substantiate that doctrine, nor are these elements "sensation centres." Since Tripier's experiments, a great number of cases are to be found in literature, showing alterations of sensation associated with lesions of the 'motor centres,' but a great number of cases are also to be found in which no such associations are to be found. In many of my experiments I have observed alterations of sensation after lesions of the 'motor centres'; also alterations of the sense of sight, and more commonly in the dog I may say that almost always the sense of sight is affected when lesions are made even on the frontal lobes. But many times also no such alteration of sensation is observed, and a "centre" to be specific must always appear at least endowed with the same faculty surely?

That the cells of the cortex do not cluster into well-defined 'centres' of innervation in the sense attached to the word 'centre' by those who teach the 'Doctrine of Localisation' is to me certain, and if all grant that the cortex contains 'centres' at all, then Brown-Séquard's well-known doctrine that centres are diffused through the whole cortical area, with possibly some 'spots' ordinarily more active;² a doctrine which he has been maintaining

¹ Dupuy. *C. R. Soc. de Biol.*, p. 274, t. 39, 1889.

² Little is known about the functions of the brain, but we can perceive that as the intellectual powers become highly developed, the various parts of

for a long time, and which is certainly supported by an immense number of observations ought to be the one accepted by those who believe the Localisation doctrine. Ferrier¹ has very clearly stated the case; "Causation is not established unless an invariable and unconditional relationship has been proved to exist between a particular lesion and a particular symptom." But if equally clearly expressed, the following statement: "In the case of the motor area it has been satisfactorily demonstrated that a destructive lesion invariably gives rise to motor paralysis, according to the position and extent of the lesion," is not proven by the experimental results and pathological observations, which I have laid before the "Association" to-night.

Before ending I beg leave to say a few words on an important question. It is apparent from the line of argument I have followed that I do not admit that the pyramidal tracts are the necessary connecting links between the cortex and the outgoing nerves of the cord. It is necessary to give some explanations. The experiments quoted of double (alternate) hemisection of the cord, but at different levels already goes some way to substantiate my theory; but there are so many cases of descending degeneration occupying the pyramidal tract alone that the demonstration of the crossed action of the motor tracts seems unshaken. Vulpian² has stated very clearly that this argument is not very strong. He gives instances in which he had seen atrophy of the white fibres of the anterior pyramids in a part of that tract, and yet above and below that atrophied portion the pyramidal fibres had a normal appearance. He gives two cases of deep lesions of the pyramids without paralysis. His statement "that voluntary incitations are not transmitted necessarily and conclusively to each side of the body by the anterior

the brain must be connected by very intricate channels of the freest communication, and as a consequence each separate part would perhaps tend to be less well fitted to answer to particular sensations or associations in a definite and inherited—that is instinctive—manner.—Darwin, *Descent of Man*, p. 68, second edition, 1888.

¹ Ferrier. "Functions of the Brain," second edition, p. 378.

² Vulpian. "Physiologie Generale and Comparée du Systeme Nerveux," p. 474 and 494.

pyramid of the opposite side" is fully borne out. A great number of experiments made by Vulpian himself, Schiff and Brown-Séquard, and myself, consisting in sections of the pyramids, and in which it was found that they are not, as Vulpian says, "exclusively and necessarily" the channels of transmission of voluntary incitations to the opposite of the body. There are a certain number of cases in literature, in which sclerosis of the bulb had destroyed one pyramid without there being a consequent paralysis.

From all I have said and all I know, I am able to state that in grey matter of the cerebro-spinal system there is no specified localisation of functions, in the sense understood by the majority of physiologists and all physicians. I believe that the grey tube element is *par excellence* the "matter" into which all the activities of the body centre; but I hold that it is inexcitable except through nerve fibres, and that it is neither 'motor' nor 'sensory'—it is potential substance. These functions of motion and sensation are the attributes of peripheric elements alone. Central grey substance simply reacts according to the nature of the impulse. Paralysis of motor activity [and of sensation] only takes place *directly* when there is destruction of the strands of fibres about to leave the cord, or leaving the cord and going to the muscles. Even when the so-called "motor cells" are destroyed or the seat of a lesion, paralysis which may appear is owing to another cause than loss of a "motor centre."¹

¹ Vulpian. A woman, about sixty years old, and in whose left leg and foot the muscles were the seat of complete fatty atrophy—probably since childhood. Nervous fibres of muscular nerves less numerous than usual, and of smaller diameter generally, showed, nevertheless, all the characteristics of normal state, and such also was the case with those of the anterior roots to which they belonged. In this case there was well pronounced asymetry of dorso-lumbar enlargement of the cord, the left half was manifestly smaller than the right. Upon section made at that level after hardening in chromic acid, Mr. Prevost, my 'interne,' found and made me see that the cells of the anterior horn of the left side had disappeared, chiefly in the external portion of that horn, where not a single one was visible ("Physiologie du Systeme Nerveux," p. 345).

If any anatomist could see as I have seen the No. viii. fibres absolutely normal in the area of Deiter's nucleus while almost all the cells were absent, he would begin to reflect on the uncertainty of the simple study of normal sections, however carefully or conscientiously carried out (Seguin, "Archives of Medicine," t. 2, vol. x., p. 261). He is speaking of Von Gudden's preparations.

The grey substance central organ of the nervous system is everywhere identical in all its portions; impressions therein arriving are differentiated by the nature of their cause—that is, they arrive under differentiated specie from peripheric elements and the diffusion of impression is more or less great, is local or general, according as the measure of the exciting activity is weak or strong; but the ‘nerve-cells’ have nothing to do with this.¹ When the grey substance is sectioned transversely it is obvious that the part below the section will only react under stimulus from the organs with which it has connection; and it can be segmented into several ‘central organs’ each independent of the other, but so long as there exists even a very small connecting link between segments of the grey substance, diffusion of impulses can take place all through, and objective phenomena become manifest as well as subjective. Now to speak of ‘localisation of functions’ in the cerebral cortex, and ‘localisation of cerebral diseases,’ appears to me only a convenient mode of expression, chiefly when used by clinicians, but devoid of any sense when used to express a physiological fact. According to Ferrier’s² statement respecting the supposed sensory function ascribed by some to ‘motor centres’: “for a single case of destructive lesion of the motor area without anæsthesia is sufficient to overthrow the apparent casual connection founded on a host of positive instances,” it is natural and logical that the same should hold good for the motor functions of the so-called ‘motor centre’ in the Rolandic area of the cortex also. If I am not mistaken, that one “single instance” means a plurality, to say the least!

My subject does not include the necessity of explaining in full how brain lesions produce objective motor symptoms, but a few words are not amiss, and with these I shall have ended. Since a motor paralysis manifest at one time dis-

¹ I need not here recall the works of Golgi and his school, and chiefly of Nausen. According to these, the cylinders-axis nerve fibres are directly connected by lateral prolongations, and reflex action takes place without passing through the cells. I know how hazardous it is to construe physiological theories upon anatomical researches however!

² Ferrier. “Functions of the Brain,” p. 378, second edition.

appears, and the cortex material lesion with which it has been connected empirically and clinically still persists, it becomes logically evident that the presence of the "centre" (the seat of the lesion) is not a necessity for the restoration of functional activity of the parts which were paralysed. Secondly, since the lesion situated in different area of the cortex is found, in a case of motor paralysis, involving the same parts of the body as in the previous observation, it is logically evident that empirically and clinically, diversely localised lesions may cause the same symptoms;¹ and if a lesion situated in a 'centre'—not the one which is said to be the "motor centre" for a given group of muscles—on being destroyed the group of muscles is found paralysed, that state of paralysis may be brought about by an influence which manifests the activities of nervous elements 'reflection,' the result of electric variation. All paralysis is the result of a reflex starting from the seat of the lesion wherever situated; in clinics, paralysis and convulsion involving a limb, and motor aphasia, are known to arise from irritations (organic, toxic, &c.) situated in the abdominal cavity sometimes. The affirmation, that in the case of hemiplegia or convulsion, epilepsy, motor aphasia, when owing to the action of a toxic agent in the system, or to the presence of worms in the bowels, do not last, while the same derangements of the motor apparatus are persistent in case of brain or 'motor centre lesion,' is so often repeated because unchallenged, besides it is irrelevant. I have given only a very few cases for the sake of brevity, but literature is rich in instances of motor derangements having disappeared, the lesion in the cortex 'motor centre' persisting. It is my intention to treat of the mechanism of paralysis from brain diseases at some future occasion; this evening I have occupied enough of the valuable time of the members of the Neurological Association.

¹ Brown-Séquard in *Lancet*, 1876-77.

ASCENDING DEGENERATIONS RESULTING FROM LESIONS OF THE SPINAL CORD IN MONKEYS.

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EXPERIMENTAL physiology has shown that the periphery of the lateral and anterior columns of the spinal cord in animals, such as rabbits, dogs, cats and monkeys, is made up of fibres which connect cells of the cord with the cerebellum, especially the vermis. The cells of Clarke's column have long been considered to be connected in this way by means of the direct cerebellar tract, and it was formerly considered by Flechsig and others that the column known as the antero-lateral tract of Gowers was a part of the ascending cerebellar tract. Bastian¹ described this connection in a case of paraplegia, and he considered the ascending antero-lateral degeneration to be a part of the direct cerebellar tract. From pathological observations (notably from a remarkable case of gun-shot injury), Gowers believes the antero-lateral column which bears his name to be connected with the transmission of painful sensations from the opposite side of the body. He states² "It has often been confounded with the cerebellar tract, and it is this which has given rise to the impression that the cerebellar tract extends further forwards than it really does. In the lumbar region, this tract lies across the lateral column, on a level with the posterior commissure from which its fibres probably come. This tract has recently been found by Bechterew to undergo development at a different period from the rest of the lateral

¹ *Med. Chr. Trans.*, 1867.

² "Diseases of the Nervous System," vol. i., Spinal Cord and Nerves.

column. It is almost certainly a sensory tract, and physiological facts seem to show that its fibres are connected with the posterior roots of the opposite side. Fibres probably pass to it through the whole length of the cord, and these are mingled together, so that the degeneration arises from a lesion of the lower part of the cord."

Edinger¹ is also of opinion that the antero-lateral tract is connected with the transmission of painful sensations from the opposite side—maintaining however, that the fibres composing the tract are derived from cells of the posterior horn and that these fibres decussate in the anterior commissure. He supports the latter statement by microscopical investigations on the spinal cords of fishes, reptiles, and amphibians, also by reference to the experimental work of Auerbach, an abstract of whose work will shortly be given in this paper. It is right to state that Auerbach himself energetically repudiates conclusions drawn from his own work by Edinger.

Since section of the posterior roots in animals produces no degeneration in the antero-lateral column, it is highly improbable that the fibres of this tract are derived from any other sources than from cells in the spinal cord, notwithstanding the recent publication of Berdez,² of which a short abstract is given below. Experiment does not seem to show

¹ Einiges von Verlauf der Gefühlsbahnen im Centralen Nerven System. Dr. L. Edinger, *Deutsche Medicinische Wochenschrift*, 1890.

² Recherches experimentales sur le trajet des fibres centripètes dans la Moëlle Epinière. Par le Dr. Berdez. *Revue Med. de la Suisse Romande*, 1892.

Berdez divided roots on one side in guinea-pigs. Some of the lesions injured also the posterior horn and posterior column. The sections were stained by Marchi's method. A scattered degeneration in both antero-lateral columns was observed above the lesion, nearly equal on the two sides, but the higher one goes the fewer the degenerated fibres seen. Bilateral degeneration of the posterior columns occurred but much more marked on the side of the lesion. The passage of fibres from one side to the other is made by the posterior part of the grey commissure. Berdez noted also the existence of degenerated descending fibres occupying the middle part of the posterior column. The existence also (but less numerous than the ascending) of descending degenerated fibres in the antero-lateral column.

In the cord of an animal so small as a guinea-pig it is very easy to produce damage, and scattered degeneration results. Moreover scattered black points are not always degenerated fibres.

The guinea-pig is not a very satisfactory animal as it is generally considered that its tracts are not so well defined as those of animals higher in the zoological scale (F. W. M.)

what cells give rise to these fibres. The fibres are large in size, they have a long course, and presumably, therefore, the cells are large. It seems that many of these fibres come from cells distributed in the lumbar region of the cord. Clarke's column has been shown experimentally by me to give origin to the fibres of the direct cerebellar tract.¹ As I shall show, this antero-lateral tract must be considered the *ventral* portion of the ascending cerebellar tract. The question thus arises—do the fibres forming it *also* spring from the cells of Clarke's column? These cells are not found in the lumbar enlargement *below* the third segment, but it is possible that the fibre may take a circuitous course downwards, and then loop upwards in the lateral column before reaching the periphery of the cord, as we find they do at their termination. Thus a lesion of the lumbar part of the cord even below the situation of Clarke's column might give rise to degeneration in the ventral cerebellar tract, or another explanation is offered by the experiments of Singer² (of which a short abstract is given below), viz., that fibres in the antero-lateral column spring from cells of the anterior cornua

¹ Bi-polar cells of the spinal cord.—MOTT, *Brain*, 1891.

² Ueber die Veränderungen am Rückenmark nach zeitweiliger Verschlussung der Bauch Aorta von J. Singer. *Akad. der Wissenschaften zu Wien, Sitzungsberichte*, 3 *Abtheilung*, 95-96, 1887.

Singer pursuing the investigations of Ehrlich and Brieger of compression of the abdominal aorta found certain changes to occur in the lumbar enlargement of the spinal cord.

Extra peritoneal compression of the aorta was made in animals just about where the left renal artery is given off. One animal was kept alive 5 weeks, the others were killed at shorter intervals of time after the operation. When the compression was made on the right side of the spine no results were obtained. The compression was maintained for one hour, and was followed by permanent paralysis and analgesia of the hinder extremities.

The resulting changes were briefly as follows:—At the end of 24-36 hours. Microscopical examination of sections of the lumbar spinal cord after suitable hardening and staining showed that the anterior cornual cells had commenced to undergo degenerative changes. The cells showed a fine granular change, and here and there around the vessels of the grey matter, escape of red corpuscles was observed.

Four days after operation there existed a well-marked granular degeneration of the anterior cornual cells, with breaking off and even absence of the processes, besides there were divers signs of change in the nucleus. The medullated fibres of the anterior horn were markedly varicose, and there were irregular clumps of myelin, and swollen axis cylinders scattered about. The white substance also showed numerous swollen axis cylinders.

Eight days after the operation. The anterior horns were smaller, the ganglion cells having disappeared. There was destruction of the medullary

in the lumbar enlargement. Testut¹ states; "the fibres constituting the cerebellar tract (which he divides into a ventral and dorsal portion) thus represent directly long anastomoses thrown between the vermis superior of the cerebellum and the different levels of the vesicular column of Clarke." That both the ventral and dorsal ascending cerebellar fibres terminate in the vermis superior, the following experimental evidence conclusively proves. See fig. 1, a diagrammatic representation, modified from Testut, of the termination of the dorsal and ventral ascending cerebellar fibres in the vermis superior.

Von Monakow,² quoted by Löwenthal,³ made an almost perfect hemisection of the spinal cord immediately below the decussation of the pyramids in a new-born rabbit, and proved that a small portion of the lateral column is continued directly up into the region of the pons. The ascending atrophy of this bundle, which is compact and of considerable size in the rabbit, is situated between the ventral extremity of the ascending root of the fifth and the lateral nucleus. He followed it further to the level of origin of the fifth pair, and considered that it was continued into the lateral part of the ribbon of Reil, and he terms it "Aberrirende Seitenstrangs Bundel."

Löwenthal's admirable description of his discovery of the course of these fibres, which I have unfortunately only recently become acquainted with, clearly shows the destination of this tract. He made a spinal lesion in the dog between the fifth and sixth pairs of cervical nerves, destroying the whole left lateral column, and he figures the course of the ascending ventral and dorsal cerebellar tracts to their termination in the vermis. The conclusions that he comes to from his experiments are as follows :

sheath of fibres in the anterior horn and certain regions of the white matter.

Three weeks after the operation. The posterior roots, spinal ganglia and posterior columns were quite normal. The anterior horns greatly diminished in size, and a disappearance of the anterior cornual cells with replacement of the nerve network by connective tissue. The anterior and posterior commissure both intact. In the anterior and lateral columns, especially the outer periphery (situation of antero-lateral tract of Gower's) are numerous degenerated fibres. Below the lumbar enlargement the spinal cord appears normal. The anterior roots, however, are partially degenerated.

¹ "Traité d'anatomie humaine," vol. ii., p. 352.

² *Archiv. fur psychiatrie*, 1883.

³ Degeneration secondaire ascendante dans le bulbe rachidien dans le pont, et dans l'étage supérieure de l'Isthme. *Revue Medicale de la Suisse Romande*, 1885.

the cerebellar bundle extends around the periphery of the spinal bulb in its lower part, from the ventral extremity of the substantia gelatinosa, up to the level of the antero-lateral nucleus. Its ultimate path is by two different courses—one part of its fibres is displaced more and more dorsally, and terminates in

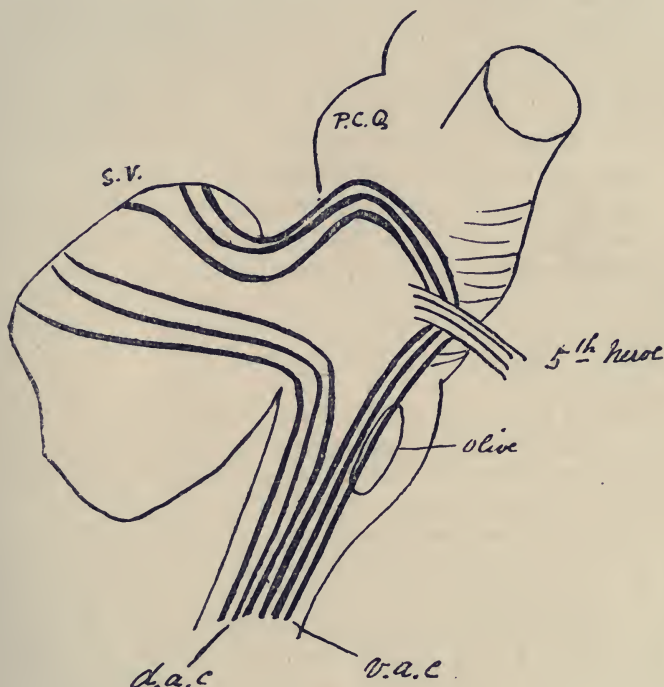


FIG. 1.

Diagrammatic representation of the course of the fibres of the ascending cerebellar tract. The ventral v.a.c. and dorsal portions d.a.c. are united in the cord and lower part of the medulla. The latter passes into the restiform body and terminates in the dorsal portion of the superior vermis s.v., the former becomes more ventral until it reaches the fifth nerve, when it forms a loop backwards to reach the posterior and outer surface of the superior peduncle. The fibres then course downwards and sweep round into the anterior portion of the superior vermis. PCQ, posterior corpora quadrigemina.

the restiform body (the dorsal portion of the direct cerebellar tract). He then gives reasons for connecting these fibres with the superior vermis.

Another portion of the cerebellar tract follows the ventro-lateral periphery of the medulla, running from behind, forwards, and always being directed more laterally (ventral portion of the

cerebellar tract). In the neighbourhood of the fifth pair of nerves it takes a deeper course, traversing the lateral region of the pons, and is covered by fibres of the middle cerebellar peduncle. A little distance behind the posterior Corpora Quadrigemina, it escapes on to the lateral furrow of the isthmus and turns towards the external periphery of the superior cerebellar peduncle, after which it runs backwards in a retrograde direction, describing outside of the said peduncle a prolonged half-turn, and comes to place itself successively on its external dorsal and dorso-internal side, which it occupies at the position where the superior cerebellar peduncle reaches the medullary velum of the cerebellum. The ultimate destination of this part of the cerebellar tract could not be elucidated. In cases where the lesion of the lateral column was limited to its dorsal half, the ascending degeneration of the cerebellar bundle could only be followed into the restiform body. The ventral portion remains intact. The fibres of the ventral portion of the cerebellar bundle only affect relations by contiguity during part of their course with the lower fillet.

Auerbach¹ destroyed the posterior part of one half the spinal cord for a considerable length, the lesion included the posterior white column, the posterior horn of grey matter and the posterior part of the lateral column. It was followed by degenerations above the lesion in the direct cerebellar tract and the antero-lateral of both sides, with degeneration of the anterior commissure. The number of the degenerated fibres of the anterior and antero-lateral columns decreases from below upwards; the fibres seem to pass into the grey matter, partly crossing at the anterior commissure. Even if the parts mentioned had been injured to a great extent in the lumbar portion of the cord (on one side only), the number of degenerated fibres is almost the same on both sides at the level of the decussation of the pyramids. The ventro-lateral cerebellar degeneration he traces up to the ventral part of the superior vermis—the dorso-lateral cerebellar tract he traces up to the dorsal portion of the superior vermis. He also makes out a third division, which originating in the lumbar cord, remains united with the ventral portion up to the level of exit of the fifth, when it enters the inner portion of the peduncle of the cerebellum (which peduncle I am unable to determine) and

¹ Zur Anatomie der aufsteigend Degeneriren den Systeme des Rückenmarks. v. Dr. Auerbach. Anatomische Anzeige, 1890; also Zur Anatomie der Vorderseitenstrangreste. *Archiv. für Pathologische Anatomie u. Physiologie* v. Virchow, v. 121, 1890.

courses towards the dentate nucleus. These fibres eventually pass from the corpus dentatum into the dorsal portion of the superior peduncle of the cerebellum. It is thus to be seen that Auerbach in a great measure confirms the observations of Löwenthal.

The previous hemisection experiments which I made upon monkeys¹ were not conducted with the view of tracing the degenerations, so much as to ascertain the physiological effects produced during life. The animals were kept a long time, and the resulting degeneration was a sclerosis. I was unable to trace the antero-lateral tract beyond the lateral nucleus in the lower part of the pons; neither was Tooth, who made hemisections in monkeys for the express purpose of tracing this degeneration, and he kept the animals alive only a short time.²

Prof. Schäfer having found the great advantage of Marchi's method in tracing degenerated fibres, made hemisections of the spinal cord in two monkeys with the express purpose of following out the resulting degenerations. He has kindly placed at my service the material, and the accompanying woodcut (fig. 2). One of his hemisections was made in the mid-dorsal region, and the other at the 11th to 12th dorsal segment. It is noteworthy that the animals, when tested as regards sensibility on the two sides, entirely corroborated (so *he* informs me) the results which I have published.³ Microphoto i. shows the ascending degeneration resulting from the latter lesion.

My own experiments were undertaken with a view of tracing the antero-lateral tract to its destination, also of ascertaining the physiological effects of its division on one side and on both sides.

The antero-lateral tract was divided, either on one side or on both sides, by means of a delicate knife, the blade of which was at right angles to a steel shaft, the

¹ Results of hemisection of the Spinal Cord in Monkeys. *Phil. Trans. Royal Soc.*, v. 183.

² When I brought the following experimental evidence before the Physiological and Neurological Societies, Dr. Tooth stated that he had altered his opinion with regard to the destination of the antero-lateral tract, and said that his observations entirely corroborated those of Löwenthal in the dog and mine in the monkey.

³ *Loc. cit.* 10.

cutting edge being in front. After exposing the spinal cord, the point of the knife was inserted into the middle of the lateral column, the edge of the knife was then pushed forward so as to divide the anterior half of one side of the cord. In one case, this was effected on the opposite side as well, so that an antero-hemisec-tion was made, that is to say, the spinal cord was completely divided in front of the central canal. The wounds were dressed antiseptically, and in all cases healed readily by first intention.¹

EXPERIMENT I.

A section of both antero-lateral columns was made in the mid-dorsal region. The left pyramidal tract was partially injured. The destruction of the anterior column was complete on the right side, and almost complete on the left. The next day after the operation the animal was tested; and, as far as I could ascertain by careful testing, with independent witnesses, no loss of painful sensation was appreciable. The animal responded as briskly to heat and pricking with a needle as before the operation; the only perceptible difference was a slight difficulty and delay in localising the presence of a clip placed on the foot or leg of the left side (that upon which the pyramidal degeneration existed). On the fourth day after the operation, the weakness of the left lower limb had passed off, and the animal behaved in all respects, as far as could be made out, like a normal monkey. It was killed at the end of three weeks with chloroform. There was most extensive degeneration on both sides in the antero-lateral columns above the lesion, also in the antero-lateral region below the lesion.

EXPERIMENT II.

Section of the right antero-lateral region of the spinal cord of the third cervical segment, and the lesion was very successful, as micro-photograph ii. shows. Half-an-

¹ The expenses of this research were partially defrayed by a Grant from the Brit. Med. Assoc.

hour after the injury, the animal had recovered consciousness, and could feel pricking and touching on both sides equally well; the pupils were equal, and both sides of the chest appeared to move normally. There was no vaso-motor paralysis, but the right arm and leg were partially paralysed. Subsequent microscopical examination showed that the cross pyramidal tract had been considerably injured.

After twenty-four hours only the *finer* movements of the hand and foot appeared much affected. Bi-lateral associated movements had already in a great measure returned on the right side. Sensation was tested as follows: the animal was prevented from seeing what I did, but when I fixed a weak clip upon the left foot (non-paralysed), it drew up its leg and removed it several times in succession. I then placed the clip upon a similar spot on the right foot, but the animal took no notice of its application. When fixed on the hand, it removed it with the teeth from both sides. When pricked with a needle, or touched with a test tube containing hot water, it gave evident signs of feeling on both sides. The animal, however, localised correctly the spot irritated on the left leg, but on the right it put down its hand, but nowhere near the place. Four days later these experiments were repeated, with the same results, and again, some few days later the animal was placed under æther, and a clip was fixed to the right foot (the side of the injury). When the animal came to, it made no attempt to remove it; whereas when fixed on the left foot, under æther, the clip was immediately removed after return of consciousness. It now feels pricking on both sides. The paralysis is much less marked, but there is some feebleness of grasp in the right hand and right foot. The animal was killed with chloroform, and the brain and spinal cord put into Müller's fluid, all precautions being taken to prevent decomposition occurring in the central parts. This animal seemed to show that localization of touch and pain is intimately connected with movement. I find that most psychologists support this view. Sully, p. 164, says in a foot-note:—"The fundamental idea here expounded that

the localization of touch-impressions and the tactual perception of space is acquired by the help of the experience of movement may be said to underly all recent attempts to trace the genesis of space perceptions. It is to be added that the German psychologists rightly emphasize the part played in the development of the perception of space by the extended surface of the skin, with its capability of yielding us at the same movement a number of locally distinct sensations."

EXPERIMENT III.

Section of the antero-lateral column on the left side, at the level of the fifth cervical segment, was made as in the previous case, care being taken to avoid, if possible, injury of the pyramidal tract. The next day the animal was tested and gave evident signs of feeling, both pricking and heat on both sides. There was slight paralysis of the arm and leg of the left side, but apparently no difficulty in localising the clip on either side. The animal alive three months after the operation, and still shows no symptoms of injury, except the scar on the skin. I have always made it a rule to test the animals before the operation, and this animal was not really a satisfactory one for the purpose of this experiment, for it did not always remove the clip nor even respond to pricking before the operation; so that no very definite inference can be drawn, although these three experiments tend to show that the inability to localise sensation is connected with injury to the posterior "*pyramidal*" portion of the lateral column. They certainly tend to negative the view that the antero-lateral tract conveys painful impulses coming from the opposite side of the body. Moreover, this view is not in accordance with the destination of this tract. Cases of compression of the cord occur in which not a single healthy fibre can be seen in the sections, and yet painful impressions, such as pricking, can be felt acutely but not localised; and cases of syringomyelia seem to show that the grey matter conducts painful sensations, as first determined experimentally by Schiff. Moreover, Singer's experiments tend to show that the grey matter conducts painful sensations.

EXPERIMENT IV.

A section of the left posterior column, the posterior horn and the posterior part of the lateral column was made between the first and second lumbar. This really corresponds to Auerbach's experiment, except that he destroyed it in a much greater length, therefore I do not attribute great importance to it. The effect of this lesion which extended for about a quarter inch was to produce weakness in the left foot, and loss of localisation to a great extent. I tested the animal in the usual manner. It felt painful sensations, but was unable to localise a clip placed on the foot, neither did it remove it upon regaining consciousness when the clip had been fixed on the foot under an anæsthetic. I am at present engaged upon further experiments of this nature. Examination of the cord shewed, however, above the lesion a scattered degeneration in both anterior and lateral columns, which gradually became less and less as the cervical region was approached. At the level of the second cervical the number of degenerated fibres in the ventral cerebellar tract of the same side were more than twice as numerous as on the opposite side to the lesion.

To revert to the degenerations resulting from Professor Schäfer's hemisections and my own experimental sections of the antero-lateral portion of the cord, it may be stated that the brain and spinal cord in these animals with the exception of Experiment III., were hardened in Müller's fluid, and the sections stained by Marchi's¹ method. The accompanying woodcut shews the degeneration above and below the lesion following a hemisection at the eleventh and twelfth dorsal. The black dots indicate the degenerated fibres. The lumbar section shews the degeneration below the lesion in the antero-lateral periphery of the cord. These descending fibres can be traced as I have shewn,² down to the extreme termination of the coccygeal portion of the cord.

¹ Small pieces of the cord, &c., are placed in a solution of 1 part 1 per cent. osmic acid, and 2 parts 3 per cent. bichromate solution for a week in a warm chamber, the temperature never exceeding 37° C. The pieces are then embedded in celloidin, and thin sections cut and mounted in canada balsam. This method is perfectly reliable, if certain precautions are taken in preparation and observation.

² *Loc. cit.*

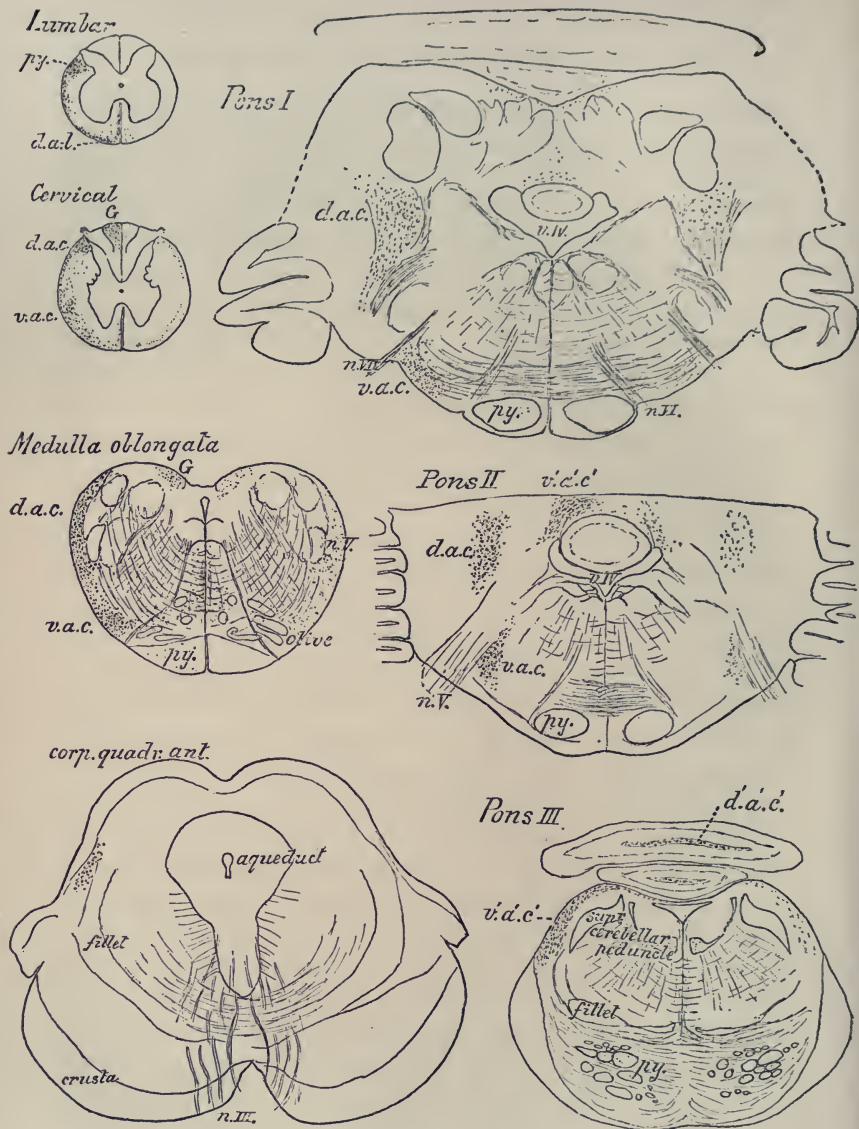


FIG. 2.—DEGENERATIONS IN THE SPINAL CORD, MEDULLA OBLONGATA, PONS VAROLII AND MESENCEPHALON OF A MONKEY FOLLOWING HEMISECTION AT THE 12TH DORSAL NERVE (E. A. S.).

The hemisection was on the left side of the cord and was complete. The section outlines drawn to a scale with a camera lucida. The degenerated fibres shown by black dots. Those in the section of the lumbar cord are descending, all the rest are ascending. *py.*, pyramidal tract; *d.a.l.*, descending antero-lateral tract; *d.a.c.*, dorso-lateral ascending cerebellar tract; *d'.a'.c'* (in Pons. III.), degeneration of fibres of this tract in the white matter of the cerebellar worm; *v.a.c.*, ventro-lateral ascending cerebellar tract; *v'.a'.c'* (in Pons II. and III.), degenerated fibres of this tract passing dorsally into the valve of Vieussens (in Pons III.) and into the white matter of the vermis (in Pons II.); *n.III.*, *n.V.*, *n.VI.*, *n.VII.*, issuing fibres of the 3rd, 5th, 6th, and 7th nerve-roots; *v.IV.*, 4th ventricle.

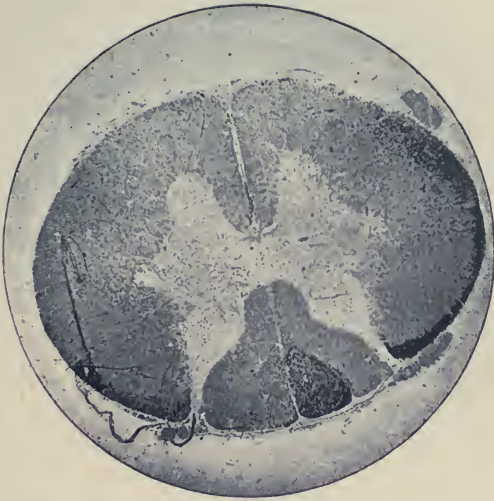
Some of them are undoubtedly long fibres, but Löwenthal¹ has shown that they do not, however, come from the cortex.

He terms this tract the *faisceau marginal*. My section of the anterior column at the third cervical segment (Experiment II.), produced a most extensive degeneration in this tract which could be traced right down to the termination of the cord. Some of the fibres seem to decussate in the anterior commissure, as the degeneration on the opposite side was more extensive low down than in the mid-dorsal region. They could be traced into the anterior cornua, possibly there they establish connections with the motor cells. Where *do* these fibres come from, if they do not come from the cortex, and therefore do not correspond to the direct pyramidal tract in man? Marchi² has answered this question by examining the cords of animals in which Luciani had produced lesions of the cerebellum, and asserts that efferent fibres from the cerebellum pass down the cord, mingled with the ascending antero-lateral and direct cerebellar tracts; so that we have the whole periphery of the anterior and lateral columns of the cord (for the most part) occupied by cerebellar fibres, some ascending and some descending. It is however possible and probable that many vertical fibres of a commissural nature to the cells of the cord at different levels exist even at the antero-lateral periphery mingled with the cerebellar fibres. With regard to ascending degeneration, nothing need be said concerning the course of the fibres of the column of Goll; this is well-known and generally accepted. Its course is shown in the figs. representing a section of the cervical region of the cord and the medulla. The ascending cerebellar tract is shown in the cervical region occupying the anterior and lateral periphery of the cord. The ventral and dorsal portions represented by v. a. c., d. a. c., are continuous, although each forms at one point a more compact and triangular area of degeneration. In micro-photograph iii., a section of the first cervical seg-

¹ *Revue Medicale de la Suisse Romande*, No. 9, 1886. La Région pyramidal de la capsule interne chez le chien et la constitution du cordon antero-laterale de la Moëlle par N. Löwenthal.

² *Ref. Neurolog. Centralblatt*, x. 8. Sullorigine e decorso dei peduncolari cerebellari e sui loro rapporti cogli altri centri nervosi pel Dr. Vittorio Marchi, Pubblicazione del R. Inst. di Studi Superiori in Firenze.

ment in Experiment II., the degeneration is almost entirely limited to the ventral portion. In the medulla, the two tracts are continuous, as shown in fig. 2, medulla oblongata; but in its upper part the two tend to become separated, as shown in pons I., the dorsal-cerebellar fibres become oblique, running backwards in the restiform body, while the ventral portion becomes more ventral and forms as shewn in micro-photographs iv. and v., islets of degeneration amidst the arciform fibres of the pons. These degenerated fibres, owing to their somewhat oblique direction outwards, appear somewhat larger than they do in the cord, and it may be noted that there are scattered degenerated fibres internal to the compact degenerated area. At the level of origin of the fifth nerve, as shown in fig. 2, pons II., the fibres become oblique, and form a loop over this nerve; and posteriorly where the isthmus of white substance connects the superior cerebellar peduncle with the cerebellum, degenerated fibres can be seen entering the anterior portion of the superior vermis (the roof nucleus of Stilling), fig. 2, v'a'c', and micro-photos vi. and vii. Still higher at pons III., just behind the posterior corpora quadrigemina, a tract of degenerated fibres is seen covering the superior peduncle of the cerebellum, and continuous with the valve of Vieussens, into which some few degenerated fibres run. Micro-photo vii. shows the triangular portion of these fibres under a high power. Still another figure shows a section of the anterior corpora quadrigemina, in which a few degenerated fibres of the fillet of the same side are shown; these I believe to be the scattered fibres which were observed internal to the compact area of degenerated ventral cerebellar fibres seen among the arciform fibres in the lower part of the pons. The dorsal cerebellar fibres could be traced from the restiform body into the dorsal portion of the superior vermis whilst the ventral portion was found as described by Löwenthal and Auerbach, to take a much more circuitous course. In the pons they leave their ventral situation, forming a loop over the fifth nerve, they are then directed obliquely upwards and backwards, on to the surface of the superior peduncle: forming a layer of fibres continuous with the valve



i.

Section of the spinal cord of 4th cervical segment. The degeneration is shown black in Goll's column, in the (direct) dorsal cerebellar and (antero-lateral) ventral cerebellar tracts in the right side. This degeneration resulted from a hemisection on the right side at 11th to 12th dorsal segment.



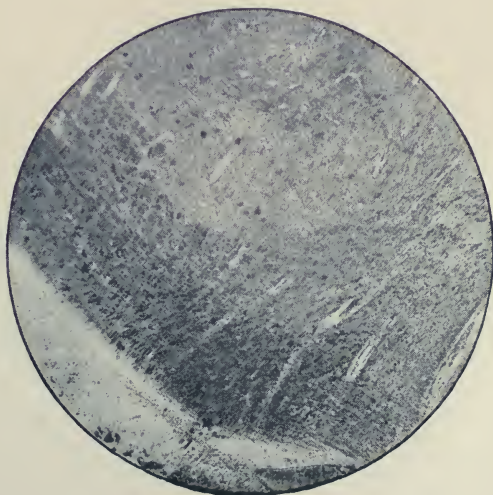
ii.

Destruction of the antero-lateral half of the cord on one side at the level of the 3rd cervical segment section near the lesion. Experiment II.



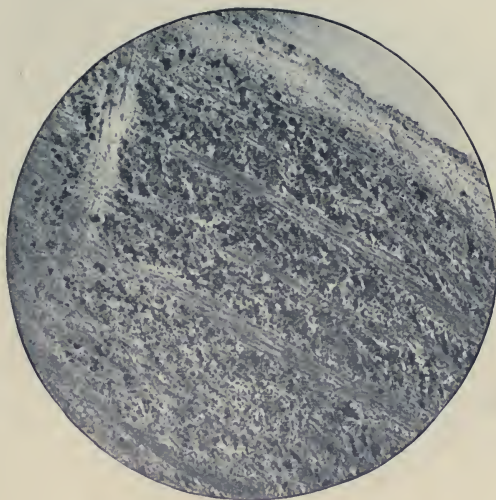
iii.

Section of the spinal cord at 1st cervical segment. The black on the left antero-lateral periphery is the ascending degeneration resulting from lesion shown in ii.



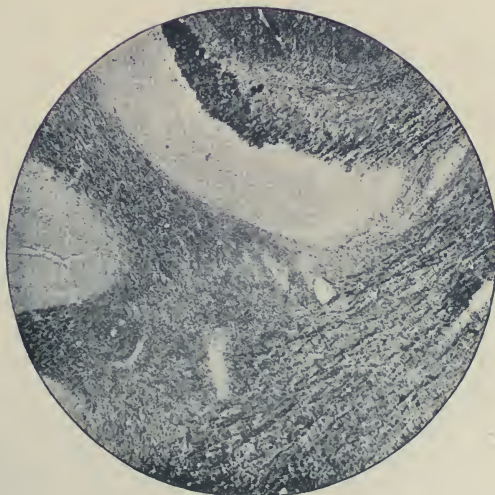
iv.

Degenerated ventral cerebellar fibres amidst the arciform fibres in the lower part of the pons, forming a compact tract between the 6th nerve and the ascending root of the 5th.



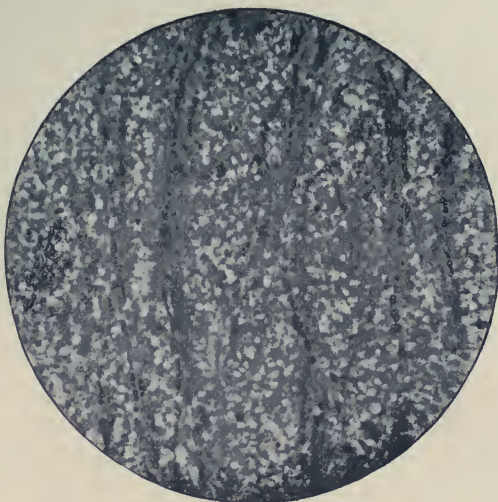
v.

The same as iv., more highly magnified. The fibres are just beginning to take an oblique direction.



vi.

The degenerated fibres, passing from the superior cerebellar peduncle into the ventral portion of the superior vermis.



vii.

The degenerated fibres, as they are passing through the isthmus into the vermis, more highly magnified than in the last vi.



viii.

The degenerated fibres of the ventral cerebellar tract shown, taking an oblique course backwards to reach the superior cerebellar peduncle. The portion shown in the photograph is v' a' c'. Pons III. Fig. 2.

of Vieussens and separated from the peduncle by a thin layer of gray matter, they then run downwards on the posterior surface of the peduncle as far as its junction with the cerebellum at the isthmus, where these degenerated fibres can be seen streaming inwards to the superior vermis. The peculiar tortuous course taken by these fibres is diagrammatically shown in fig. 1. On looking over my old preparations, prepared by Weigert's method, I can now follow in them the course of the degeneration of this tract,¹ although I previously stated that I was unable to trace the degeneration further than the lower border of the pons.

Summary.—The peripheral portion of the anterior and lateral columns consists in great part of ascending and descending cerebellar fibres. The former may be divided into a ventral and a dorsal portion, which may be termed the *ventral and dorsal ascending cerebellar tract*, instead of antero-lateral and direct cerebellar. The ventral portion may be completely divided in monkeys apparently without producing analgesia. The ascending cerebellar tract, consisting of ventral and dorsal portions forms a connecting bridge between the superior vermis of the cerebellum and cells in the cord. The dorsal portion connects the cells of Clarke's column with the dorsal portion of the superior vermis. The ventral portion connects certain cells of the cord with the ventral portion of the superior vermis. Section of the antero-lateral column in the cervical region produces a much denser degeneration amidst the arciform fibres than can be accounted for by the slight injury to the direct cerebellar fibres; the *very extensive tract* of degeneration corresponding to Gower's antero-lateral tract, which, with Löwenthal, I agree should be called ventral cerebellar, has been traced in the monkey to the superior vermis by way of the superior cerebellar peduncle, forming in its course a remarkable loop over the 5th nerve, as shown in fig. 1. Scattered fibres belonging to the fillet can be traced as far as the anterior quadrigeminal body of the same side.

¹ The Micro-photos are, for the most part, taken from specimens made from the cord and brain of Case II., section of the antero-lateral column, at the third cervical segment. *Loc. cit.* Results of hemisection of the spinal cord in monkeys.

THE CHANGES IN THE OPTIC TRACTS AND CHIASMA, IN A CASE OF UNILATERAL OPTIC ATROPHY.

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IT is now allowed by all observers—with the exception of Michel—that a semi-decussation of the optic nerve fibres occurs at the chiasma, but as to the exact relation of the crossed and uncrossed fibres in the optic tracts and in the chiasma, considerable diversity of opinion exists.

On looking over the records of the microscopical examination of the optic tracts and chiasma, in cases in which unilateral blindness had existed for some time previous to death, one finds that the results vary. In many of the cases reported no definite *bundles* of degenerated fibres were found in the optic tracts. In two cases of unilateral optic atrophy, Gowers¹ found that there was a little excess of connective tissue in both optic tracts. The microscopical changes were nearly equally distributed through both tracts but the tract, on the side opposite the affected nerve was rather smaller than the other.

Baumgarten² and Burdach³ report cases in which tracts of degeneration were traced for a short distance only beyond the chiasma.

In some of the cases recorded, bundles of degenerated fibres have been traced in the optic tracts.

Thus, Siemerling⁴ found, in a case of unilateral blind-

¹ GOWERS, *Centralbl. f. die Med. Wissensch.*, 1878, No. 31, S. 562, and *Medical Ophthalmoscopy*, p. 118, third edition.

² BAUMGARTEN. *Centralbl. f. die Med. Wissensch.*, 1878, No. 31.

³ BURDACH v. *Graefe's Archiv. f. Opth.*, xxix., 3 S. 135.

⁴ SIEMERLING v. *Archiv f. Psychiatrie*, xix., S. 401.

ness, that the uncrossed bundle of fibres occupied a central position in the tract, but the boundary between crossed and uncrossed fibres was not sharply defined.

Purtscher¹ found a degenerated part in the centre of the tract on the same side as the blindness, and at the periphery of the opposite tract. v. Gudden² found in a case of total atrophy of one optic nerve, that the uncrossed fibres were more in the upper part of the tract, the crossed fibres more at the lower margin. Marchand³ states that the uncrossed fibres were situated more upwards and inwards, the crossed fibres below and inwards—in a case of unilateral optic atrophy.

All previous observations show, that there is considerable difficulty in tracing degeneration beyond the chiasma. In some cases the results seem to point to a mixing of the crossed and uncrossed fibres in the optic tracts. In other cases, the crossed and uncrossed fibres had a more or less isolated course, in distinct bundles, but in none were these bundles sharply separated, and the exact relation of the crossed and uncrossed fibres, has varied somewhat in different cases.

Through the kindness of the late Dr. Ross, some time ago I had the opportunity of making a microscopical examination of the optic tracts and chiasma, in a case of complete unilateral blindness.

The following is an abstract of the notes which I took on the case during life:—

Complete loss of vision, right eye: atrophy of R. optic disc: left eye normal: cardiac dilatation: œdema and ascites. History of sudden onset of blindness in r. eye, after an attack of rheumatism, four years previous to death. Autopsy. Cardiac dilatation: old perihepatitis: cirrhosis of liver; ascites: atrophy of r. optic nerve.

Hannah T., age 56, was admitted as an in-patient at the Manchester Royal Infirmary, under the care of Dr. Ross, on December 30th, 1890.

¹ PURTSCHER v. *Graefe's Archiv. f. Ophth.*, xxvi., 2, S. 191.

² v. GUDDEN v. *Graefe's Archiv. f. Ophth.*, xxv., 4, S. 237.

³ MARCHAND v. *Graefe's Archiv. f. Ophth.*, xxviii., 2, S. 63.

Previous History.—About eight years before admission, the patient suffered from an attack of rheumatic fever. About four years before admission, she had a second attack. One month after her recovery from the second attack of rheumatic fever, she suddenly became blind in the right eye [?embolism of central artery of retina]. At the time of onset of the blindness, she was sitting on the doorstep exposed to the heat of the sun. The right eye suddenly became almost blind, and began to “water;” but there was no pain. Vision became more and more impaired, and in the course of a week, the right eye became totally blind. The left eye was not affected, and there were no other symptoms of lesion of the nervous system.

Soon after the onset of blindness in the right eye, she suffered from bronchitis and pleurisy. In the summer of 1890 she was an in-patient at the Manchester Royal Infirmary, suffering from dilated heart (probably alcoholic).

Present State:—

December 31st, 1890.—Patient is an exceedingly stout woman. Marked orthopnœa. Purpuric spots scattered over the limbs and trunk. Slight œdema of the legs. Marked ascites.

Circulatory System.—Marked dyspnœa and palpitation; pulse 75, irregular in force and rhythm, feeble and easily compressible. Well marked carotid pulsation. Also pulsation in the external jugular veins, which ceases on pressure on the vein just above the clavicle. Heart: apex impulse difficult to localise on account of obesity. On percussion, apex in the sixth space, four inches from the left edge of the sternum; deep cardiac dulness begins at the third left cartilage and superficial dulness at the fourth. Deep dulness extends half-an-inch to the right of the right edge of the sternum. A soft systolic murmur is heard at the apex, also heard at the aortic region.

Respiratory System.—Cough troublesome. Abundant muco-purulent expectoration. Respirations 36 per minute; râles heard all over the chest.

The abdomen is greatly distended with fluid, which prevents the area of the liver dulness from being mapped out.

Urine scanty, high coloured, 1020, acid, no albumen, no sugar.

Eyes.—Slight external strabismus of the right eye. Pupils $R > L$. No perception of light in the right eye.

Ophthalmoscopic Examination.—Media of both eyes normal. *Right eye.*—Well-marked atrophy of the optic disc. Disc of a dead white colour, margins well-defined. No irregularities of the margin. Slight cupping of the disc. Vessels somewhat smaller than those of left fundus (but the difference is only slight). No white lines along the vessels. No other abnormalities detected in the right fundus.

Left eye.—Vision, field of vision, and fundus normal.

No paralysis of ocular muscles.

No signs of any lesion of the nervous system.

Briefly, the subsequent history was as follows:—

The patient's abdomen was tapped on January 3rd, 1891, and a large quantity of ascitic fluid withdrawn. Soon afterwards, œdema appeared in the right arm, and then in the left. The dyspnœa and cyanosis became more marked. The œdema in the legs increased, and fluid soon re-accumulated in the peritoneal cavity. From January 11th to the time of her death on January 23rd, she suffered from considerable pain in the abdomen.

Post-mortem Examination.—Briefly stated the following were the results of the autopsy:—

Abdominal cavity contained a large quantity of yellowish slightly turbid serum. There were a large number of recent peritonitic adhesions.

Heart, greatly enlarged; weight 32 ozs. Cavities much distended, and orifices dilated. With the exception of some very slight irregularities on the margins of the mitral valves, the valves of the heart were normal. Ventricular walls pale, and showed distinct signs of fatty degeneration.

Liver: Capsule of a dead white colour, thick and firm on section. Liver cirrhotic.

Spleen a little enlarged, capsule white and firm. Kidneys congested.

Dura mater adherent to skull cap. The base of the skull and the region of each optic foramen, normal.

The right optic nerve, from the eye-ball to the chiasma, is markedly atrophied; it is much smaller than the left, which is of normal size. At a point eight millimetres in front of the optic chiasma, the greatest diameter of the right nerve is three millimetres, the greatest diameter of the left nerve, at a corresponding point, is five millimetres. The right optic nerve has a greyish colour, the left is of the normal dead white colour. There are no signs of meningitis, tumour, or other abnormality about the right optic foramen. Nothing abnormal in the right orbit, nor in the region of the chiasma, or optic tracts. The orbital portion of both optic nerves, and the posterior half of the right eye-ball, were removed for microscopical examination. To the naked eye, the posterior part of the eye-ball appeared normal. The basal ganglia of the brain, crura, pons, and medulla, with optic tracts and chiasma attached, were separated in one piece from the cerebral and cerebellar hemispheres, and placed in Müller's fluid. The external and internal geniculate bodies, the corpora quadrigemina, optic thalami, internal capsules, occipital lobes, supra marginal convolutions, and all other portions of the cerebral hemispheres, pons, medulla, and cerebellum, were normal to the naked eye.

Microscopical Examination.—After hardening in Müller's fluid, the various parts were embedded in celloidin, and the sections stained according to Weigert's hæmatoxylin method. Sections of the optic nerves were made about one centimetre behind the eye-balls, again midway between the eyeballs and the optic foramina. Also the whole of the nerves, from the optic foramina to the chiasma, were cut into sections. The whole of the chiasma was cut into vertical transverse sections. The whole of the right optic tract, and the greater part of the left tract, were cut into transverse sections.

Optic Nerves.—About one centimetre behind the eye-ball, the sections of the right optic nerve are smaller than those of the left. The right nerve is ovoid in shape, and its greatest diameter 2 mm.; the shortest $1\frac{1}{2}$ mm. (excluding the sheath of the nerve, which is separated from it by a comparatively wide interval). Sections of the left nerve are roughly circular; diameter 3 mm.; the nerve is closely surrounded by its sheath.

In the right nerve, the connective tissue trabeculae are greatly increased; the nerve fibres are *almost absent*; only a very few minute bundles of fibres remaining near centre of the nerve, but towards the inferior surface (stained black in the Weigert's specimens). The remaining portions of the nerve consist of granular looking connective tissue (stained yellowish brown in the Weigert's specimens), with an occasional nerve fibre (see diagram 1). The nerve is well supplied with large blood vessels the external coats of which show an increased amount of fibrous tissue. Sections of the nerve, midway between the eye-ball and the optic foramen, and also close to the chiasma, present a similar appearance. Immediately in front of the chiasma, at the inferior (basal) part of the right nerve is a small bundle of nerve fibres, running transversely, and parallel to the surface. These fibres are seen in longitudinal section; the bundle is broadest at the inner side of the nerve, and gradually diminishes to the outer side. The above-mentioned minute bundles of nerve-fibres seen in transverse section, near the centre of the nerve in its orbital portion, gradually take up a position towards the outer side of the nerve, close to the chiasma (see diagram 3). The left optic nerve is normal (see diagrams 2 and 4).

Chiasma.—The whole of the chiasma was cut into vertical transverse sections. In the first (most anterior) sections, a very small bundle of nerve fibres was seen at the inferior surface of the *right half*, corresponding to the fibres seen on the inferior surface of the right optic nerve, immediately in front of the chiasma. The fibres were seen in longitudinal or oblique section, and ran parallel to the surface. The bundle was broadest at the median side, and blended here with the nerve fibres of the left half of the chiasma; towards the outer side of the right half of the chiasma the bundle gradually diminished in size. To the outer side of the right half of the chiasma, a very small bundle of nerve fibres was seen in transverse section. The whole of the right half of the chiasma, with the exception of the fibres just mentioned, consisted of connective tissue (similar to that of the right optic nerve), without nerve fibres. The *left half* of the

chiasma consisted of normal nerve fibres, in the first sections (see diagram 5). Very soon, however, a narrow streak of degeneration (connective tissue, from which the nerve fibres had disappeared) was seen at the inner (median) part of the inferior surface of the left half of the chiasma. This streak of degeneration commenced at the middle line, on the inferior surface, and passed outwards and a little upwards (see diagram 6). Passing backwards, the streak of degeneration gradually increased in size (see diagram 7); and then, in the posterior half of the chiasma, gradually diminished. The streak of degeneration was always broadest at the median side, and gradually diminishing towards the outer side of the left half of the chiasma.

In the *right half* of the chiasma, the nerve fibres gradually increased in number on the inferior surface, and the area of degeneration gradually diminished and became limited to superior surface (see diagram 7). After the first few sections, the nerve fibres were seen in transverse section, at the inferior and outer side of the right half of chiasma, whilst those towards the median part were seen in longitudinal and oblique section, passing from the left to the right half.

In a few of the most posterior sections, a band of fine nerve fibres was still seen, running transversely between the two halves of the chiasma; and it was difficult to make out any tracts of degeneration in either half of the chiasma.

Optic Tracts: Naked eye appearance of hardened specimen.—On division of the tracts at right angles to their long axis, the *left* tract was seen to be decidedly smaller than the right. The greatest diameter of the right tract, a short distance behind the chiasma, was 4 mm.; its shortest diameter 2 mm. The greatest diameter of the left tract was about $3\frac{1}{2}$ mm., its shortest $1\frac{1}{2}$ mm. Also, for a short distance behind the chiasma, the inferior (basal) surface of the left tract was more flattened than that of the right, especially at the inner (median) side.

To the naked eye, the right tract (on division at right angles to its long axis), presented a pale coloured area in the centre. This pale area was ovoid in shape, the broad end

being to the median side (see diagram 8). On division of the left tract, no such pale coloured area was found in its centre. The whole of the cut surface of the left tract had a uniform appearance, with the exception of a very slight pale coloured rim just at the outer margin (see diagram 9). The pale area in the centre of the right tract, was seen each time the tract was cut, almost up to the point at which it divides. It may be here mentioned, that both tracts had undergone exactly the same treatment. The whole of the crura, pons, medulla, and basal ganglia, with the tracts and chiasma attached, had been placed together in Müller's fluid, and the tracts were not cut until the specimens were thoroughly hardened.

In sections stained according to Weigert's method, the right tract presented, to the naked eye, a pale brownish yellow, ovoid area in the centre, corresponding to that seen on the cut surface of the tract (see diagram 10). This pale area was present in all the sections, both in thin, as well as in very thick sections. Microscopical examination revealed an ovoid central area (corresponding to that seen by the naked eye), in which there was an excess of connective tissue (stained brownish yellow in the Weigert's specimens). In this area, the nerve fibres were less numerous, and more widely separated than at the periphery of the tract. Also the fibres were very much smaller than those in the peripheral zone (? atrophied fibres). The increased connective tissue in the central area had a granular appearance, and was well supplied with blood vessels. In section stained with aniline blue black, this central area under a low power, appeared slightly darker than the rest of the section. The area in which the connective tissue was increased came nearer the surface of the tract at the median side than elsewhere. Examination of the left optic tract, stained according to Weigert's method, failed to reveal any area of degeneration in its centre. At the centre of the left tract, the nerve fibres were quite as large, and as numerous, as at the periphery; and there was no excess of connective tissue. But at the inner half of the inferior surface, was an area, in which the nerve fibres were exceedingly scanty, and separated by a large amount of connective tissue (see diagram

11b). Extending along the inferior surface and outer side of the tract, was a very much narrower zone, in which there was a slight excess of connective tissue. The changes in the tracts just described, were seen in all the sections, from the chiasma almost up to the external geniculate bodies—up to the point at which the fibres separate into two bundles. The central area (in which the connective tissue was increased) was situated, somewhat more to the median side, however, as the right tract approached the external geniculate body; but otherwise the degenerated portion occupied almost the same position in all the sections, from near the chiasma to the most posterior part. The external geniculate bodies, and the anterior and posterior tubercles, of the corpora quadrigemina, appeared normal on section.

Judging from the changes found in the case just recorded, it would appear that the optic nerve-fibres, on crossing at the chiasma pass to the inferior surface of the opposite half. In the tracts, it would appear that the uncrossed fibres take a central position; and that crossed fibres lie at the inner half of the inferior surface, and, perhaps, along the periphery, at the outer half of the inferior surface, and at the outer side of the tract.

There were no signs of degeneration at the upper surface of the tract, on the side opposite to the atrophied optic nerve.

When one remembers, that the optic tracts are not simply composed of crossed and uncrossed fibres, but contain also a large number of fibres, passing from one tract to the other (posterior commissural fibres—Meynert and Gudden), one would not expect the degenerated part in the one tract, to be the EXACT counterpart of that in the other. Still it is somewhat strange, that in the above case and in the other cases recorded, the changes in one tract do not approach more to the counterpart of those in the other.

The facts, however, of the above case seemed worthy of record: that in a case of atrophy of the *right* optic nerve, the following changes were found:—

Chiasma.—*Right half* of the chiasma degenerated (anteriorly). The degeneration became more and more limited

to the superior surface of the right half, and gradually diminished (posteriorly). In the left half of the chiasma anteriorly, a narrow streak of degeneration was seen, commencing at the inferior surface, near the middle line, and running obliquely outwards and a little upwards. This area of degeneration soon increased in size, and then in the posterior part of the chiasma gradually diminished.

The *left* (crossed) *optic tract* was distinctly smaller than the *right*. Naked eye examination revealed a pale area in the centre of the *right tract*. On microscopical examination, in this central area there was an increase of connective tissue, and a diminution of nerve fibres.

In the *left tract* there was a small area at the inner half of the inferior surface, in which the connective tissue was increased, and the nerve-fibres scanty. Also a similar, but much narrower zone, extending along the periphery at the inferior surface, and at the outer side of the tract.

Description of Diagrams. Sections of Optic Nerves, Chiasma and Tracts, in a case of Unilateral Optic Atrophy.

All the diagrams represent sections, stained according to Weigert's method, except 8 and 9. The dark portions (dots and lines) represent healthy nerve-fibres (stained black). The lighter portions represent degenerated parts (stained brownish yellow). The numbers are placed at the *inferior* surface of the diagram of each section.

1.—Right optic nerve and sheath, 1 cm. behind the eye-ball, $\times 5$ diameters.

2.—Left optic nerve, at a corresponding point, $\times 5$ diameters.

3.—Right optic nerve, immediately in front of the optic chiasma, $\times 5$ diameters.

4.—Left optic nerve, immediately in front of the optic chiasma, $\times 5$ diameters.

5.—Section (transverse vertical) of most anterior part of chiasma, $\times 5$ diameters.

6.—Section of chiasma a little more posteriorly.

7.—Section of chiasma about the centre.

8.—Naked eye appearance of surface of right optic tract, on transverse section, $\times 2$ diameters.

9.—Naked eye appearance of surface of left optic tract on transverse section, $\times 2$ diameters.

10.—Transverse section of right optic tract (Weigert's stain), $\times 10$ diameters; (a) = degenerated part.

11.—Transverse section of left optic tract (Weigert's stain), $\times 10$ diameters; (b) = degenerated part.



EXPLANATION OF ILLUSTRATIONS.

FIG. A. To show obliterated vessels—at *x* and other parts. Transverse section of Ulnar N. at wrist (alum-carminé).

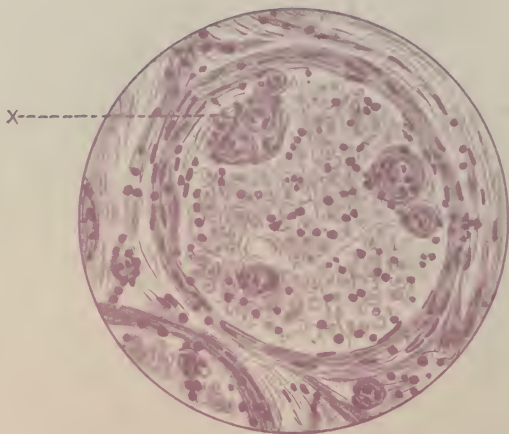


Fig A

FIG. B. Transverse section of an artery lying outside a funiculus of Median N. at wrist. To show the thickening of internal coat. At *x* the elastic layer of this coat (alum-carminé, $\times 360$).

FIG. C. Transverse section of an artery lying outside a funiculus of Ulnar N. at wrist. To show complete occlusion (alum-carminé, $\times 480$).

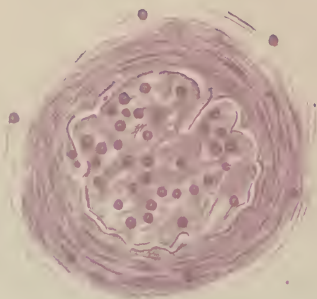


Fig C



Fig B

ON CERTAIN MICROSCOPICAL CHANGES IN THE NERVES OF THE LIMBS IN CASES OF GENERAL PARALYSIS OF THE INSANE.

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WE examined microscopically nerves from the limbs of ten patients, taken without selection, with the object of ascertaining any morbid condition which might be present. Of these, nine were undoubtedly general paralytics, judged clinically and pathologically; one case did not present the pathological appearances commonly found in the brain and surrounding membranes in general paralysis, but such clinical symptoms as presented themselves were compatible with that disorder, and the peripheral nerves showed in marked degree the changes exhibited by the nerves in the other undoubted cases.

The ages of six of the patients at the time of death ranged between 41 and 49 years; two were 36 and 37 respectively; one 29, and one 58.

The duration of the disease (in years) was, in two cases, 4, in two others 2, in one 6, in another 3. Two others died 18 and 14 months respectively from the time at which symptoms were first observed. Lastly, in two cases symptoms of insanity had been noticed for four months only—this period including observations made outside and inside the asylum; but in these the real duration of disease cannot be even approximately given.

Mention is made of atheroma—existing either in the vessels at the base of the brain or in the aorta—in six cases.

It is noteworthy that no evidence of *syphilis*—either clinical or pathological—was forthcoming in any of the cases. Apart from the examination of the patients, inquiries were made of the relatives, in the usual way; no history of specific disorder was obtained, however: the usual doubt of course attaches to the replies. Nevertheless, the fact is interesting, and, viewed in the light of the morbid change shortly to be described, significant—that no evidence of syphilis was obtainable in any single case.

The nerves examined were the median, ulnar, and posterior tibial, on each side and at the following sites: median and ulnar at the wrist-joint and in the middle of the arm, posterior tibial behind the internal malleolus, a short distance above the point of division. At each site a portion of nerve about $2\frac{1}{2}$ in. long was removed; this was hardened in Müller's fluid, and subsequently in a solution of bichromate of potash (four per cent.), or in Ehrlitzki's fluid. Certain of the nerves were hardened and stained in osmic acid (one per cent.) Sections were cut in transverse and longitudinal directions. For staining we commonly employed anilin blue-black (0.25 per cent. aqueous sol.) alone or in combination with hæmatoxylin (the combination being particularly adapted to the staining of axis-cylinders and connective tissue); alum-carmine, and the Weigert-Pal method. Portions of nerves were also cut fresh (æther-frozen) and stained with anilin blue-black and with osmic acid.

Our attention was attracted to the following morbid conditions: (a) indications of chronic inflammation and degeneration, (b) obliteration of blood-vessels.

(a.) These indications were as follows:—(1) With nuclear dyes:

Increase in the amount of connective tissue, especially affecting the perineurium and its prolongations, and the endoneurium. Here and there blocks or masses of connective tissue. Small round cells in numbers throughout the bundles and especially numerous about the blood-vessels; here and there masses composed of such cells. Nuclei prominent and numerous in many bundles.

Fat increased in amount between the funiculi.

Blood-vessels numerous, both without and within the bundles; many of large size, and very many full of corpuscles (the congested vessels are a prominent feature). Thickening of the walls of the vessels, and increase of nuclei upon them.

Alteration of nerve fibres. Marked variation in size was exhibited; by the side of quite small fibres were others, very large and irregular in shape (these were apparently swollen), and often with homogeneous, structureless aspect. Numbers of small fibres also visible. Granules of myelin frequently met with, about small heaps of such granules were collections of round cells. Much variation in the state of the axis-cylinder was observable: the stained area representing it was often of considerable size, whilst the degree of staining was faint; sometimes a still greater increase was apparent, and then the transverse section of the nerve—with the exception of a narrow ring of myelin outside—was represented by a patch, faintly stained, and uniform in tint: an appearance indicative of disease of the axis-cylinder alone, or such combined with morbid change in the quality of the myelin, of a kind permitting of absorption of nuclear dyes. In rare instances no surrounding ring of myelin was visible. In many of the most altered nerves there was no indication of axis-cylinder.

(2) With the Weigert-Pal method:

Whereas the fibres of a healthy nerve are stained by this method with, at any rate, an approach to uniformity, those of the nerves under consideration reacted to it very unequally. Alongside broad, well-stained rings of myelin, enclosing a small unstained area (healthy fibres) were others, narrow, and faintly-stained, limiting an unstained patch of comparatively large diameter. The method brought out also the irregular contour of many of the nerve-fibres, the number of small fibres (in many bundles), and lastly, the granular, broken-up state of the myelin. The sections stained by this method were checked by like ones treated with nuclear dyes, they were also compared with similarly-stained sections from a healthy nerve. Unless such com-

parison be made, misleading results are likely to be obtained, in the case of nerves stained according to the Weigert-Pal process.

To the above summary of the signs of a chronic neuritis¹ a few remarks may be added. These signs were far more marked in what may be called the "low" nerves (those at wrist and ankle) than in the "high" nerves (those in arm); in the latter, indeed, a vascular congestion, with an increase in thickness of the walls of the vessels, and an accumulation of round cells about them, often constituted the most prominent morbid features.

In the case of the "low" nerves, we observed that sections taken from the peripheral extremity of a piece of nerve between two and three inches long not infrequently showed the signs of disease described above in much greater degree than did sections from the central extremity.

Corresponding portions of nerves—whether at arm, wrist or ankle—were found to be very unequally affected.

Lastly, there was by no means a dearth of healthy nerve fibres, notwithstanding that so many fibres showed the morbid conditions above enumerated.

The state of the spinal nerves in cases of general paralysis of the insane has been very little inquired into, so far as we can gather.² Déjerine³ describes morbid appearances which he met with in one case: the myelin and axis-cylinders were considerably affected. Bevan Lewis,⁴ many years since, dealt with the histology of the great sciatic nerve in cases of general paralysis.

(b.) Obliteration of blood-vessels.

In addition to the vascular changes above described the following morbid appearances pointing, in our opinion, to obliteration of blood-vessels, were present in eight out of ten cases. Patches, circular or oval in shape, varying in size, and composed of organised tissue were seen both within

¹ Present in greater or less degree in nine out of ten cases.

² Mendel (*Die progressive Paralyse der Irren*, 1880) refers to the lack of information upon the subject. He himself does not describe any morbid condition in the spinal nerves. We have not met with any more recent publication containing reference to such.

³ *Archiv. de Physiol.*, 1876.

⁴ *West Riding Asylum Reports*, vol. v., 1875.

and without the funiculi—chiefly within, and more particularly in proximity to the perineurial sheath (see illustration A). Stains such as logwood and alum-carmine bring out the connective tissue of which these patches are composed, and often clearly show its concentric arrangement. It should, however, be noted that although the areae referred to frequently present a “formed” or “organised” appearance, as illustrated in the diagram, they sometimes look homogeneous and almost structureless, being uniformly and faintly stained (in some cases scarcely at all stained) and presenting hardly a sign of their actual composition. One is inclined to suppose that the latter appearances indicate a later stage of disease than the former; that, in fact, they point to a retrograde change in the connective tissue of which the patches are composed.

Apart from the suggestiveness of the appearances just described, reasons may be adduced in favour of the view that they are indicative of an obliteration of the blood-vessels.

1. In many instances the mass of organised tissue has around it a ring, or two or more rings of nuclei having precisely the appearance of the nuclei of muscular fibre-cells, and another outermost covering composed of connective tissue, and having the appearance of an adventitial sheath.

2. The circular patch of connective tissue sometimes lies in a prolongation inwards of the perineurium, next to a small blood-vessel, although the diameter of the former is larger than that of the latter, and the two structures might be considered very different, on superficial consideration, much the same description is applicable to the coverings of the one and the walls of the other. If we suppose that, in the first instance, two vessels of equal size lay next to each other, and that thrombosis took place in one of them, the appearances just described are explained, including the disparity of size, for in the course of thrombosis increase in thickness of the vessel-wall is likely enough to occur.

3. Blood-vessels may be seen in which a clot is surrounded by a thickened *tunica intima*: the lumen is evidently diminished in size. Or there may be considerable thickening

of the internal coat, the opposite sides of the vessel nearly meeting in the middle line (see drawing B). In those instances obliteration appears to be in progress.

4. Structures are visible, composed of a central organised mass and three circularly-disposed coverings (external, connective tissue; middle, muscular; internal, elastic); these are undoubtedly vessels which have undergone obliteration (see illustration C).

5. Between and parallel to the nerve fibres in some *longitudinal* sections capillaries may be seen, presenting at intervals in their course short fusiform dilatations. At these places the lumen of the vessel is obliterated; only a plug of connective tissue presents itself, whereas blood-corpuscles are clearly visible within the vessel at other parts, where its diameter is uniform. (Here and there along the course of the capillary its walls are distinctly thickened, so that the lumen is encroached upon.) It seems probable that the patches under consideration represent transverse sections of these fusiform swellings.

Proceeding, then, upon the view that occlusion of vessels is the morbid condition present, it may be further noted that the vessels chiefly affected are small ones—capillaries, and those a little larger, the walls of which show some muscular fibre. Considerably larger ones may be involved, as the drawing C shows; here the obliterated vessel is seen to have a well-marked internal elastic layer. We were not fortunate enough to obtain a longitudinal section showing plugging of one of these large vessels.

The appearance indicated at *x* in diagram A is frequently met with; possibly it is due to the implication of two vessels in juxtaposition.

In each of the eight cases which showed the obliteration of vessels the morbid condition was found to be present in about one-half of the nerves examined; thus, out of ten nerves four or five presented it. The “low” nerves were affected in this way far more frequently than the “high” ones. Corresponding parts of corresponding nerves were found to be very unequally implicated: in some instances, in fact, sections taken from a nerve on one side showed no

morbid condition of the vessels, whereas those from the same part (approximately) of the corresponding nerve on the opposite side of the body contained great numbers of obliterated vessels. In a given section the number of funiculi showing plugged vessels varied considerably; thus, in one section, twenty out of twenty-seven funiculi contained them; in another, composed of almost as many bundles, only two contained plugs. The number of plugs contained in one bundle varied from one to ten.

It is noteworthy that whilst the two morbid conditions here described (chronic neuritis and obliteration of vessels) commonly occurred together, several instances were observed in which vessels were obliterated within a funiculus whilst the surrounding nerve-fibres and connective tissue appeared, so far as one could determine, healthy. This fact is incompatible with the view that the obliteration of the vessels is a result of the surrounding inflammation in all cases.

In this connection may be mentioned an explanation of the mode of production of the condition in question put forward by Mendel:¹ according to him the adventitial spaces of the small cerebral vessels are filled with white and red corpuscles and masses of pigment; at some parts the collection is greater, and at these the vessel is compressed to such an extent that the lumen may be obliterated and thrombosis result. In the sections examined by us there was no evidence at all that the plugging of the vessels was due to compression by products of exudation.

Obliteration of the vessels of the cortex cerebri in cases of general paralysis has been described by various writers,² but hitherto we have not found any description of or reference to a similar state of the vessels of the peripheral nerves in that disease. In the case of the cerebral vessels, "This obliteration has usually been attributed to proliferation, sclerosis, and contraction of the vessels, or of their outer tunic, or occurring external to it; the newly-formed material constricting the vessels here and there, either occluding them

¹ *Op. cit.*, the Author refers to *cerebral* blood-vessels; those of the peripheral nerves are not mentioned.

² See Mickle, "General Paralysis of the Insane," second edition. .

directly, or by the intermediation of thrombosis. . . .” (Mickle). In the case of the nerves, although, as said, there was no evidence that the vessels were constricted by newly-formed material, there was no doubt of the fact many occluded vessels passed through tissues affected by chronic inflammation; and the occlusion was possibly due to spread of the morbid process to the vessel-wall. But in several instances, as stated above, vessels completely plugged could be seen lying in healthy-looking tissue. It may be argued that even here the real cause of thrombosis was probably inflammation around the blood-vessel, the changes attending which might have been discovered had the sections been taken from a lower part of the nerve. This theory, however, does not account for the disproportionate degree in which the internal coat is affected, in vessels large enough to show an elastic layer at the outer limit of the *tunica intima* (see the sketch, B). In these the increased thickness of the internal coat is often remarkable, any similar alteration of the other coats being of a subordinate kind. The intima is generally and equally affected; its appearance is that of ordinary connective-tissue (the cellular elements are, however, more prominent than in well-formed connective tissue); there is no evidence of fatty change.

The presence, as in the case of the vessels just referred to, of a line of demarcation between the internal and muscular coats—the elastic layer—renders it easy to localise a morbid change in respect to these coats. But in the case of vessels rather larger in size than capillaries (the actual capillaries need not, for obvious reasons, be considered), with walls composed of internal, thin muscular, and external coats, the degree in which a particular coat participates in a morbid change cannot be stated with any precision, since the coats are not clearly marked off from each other. Many such vessels, with thickened walls and narrowed bores, could be seen in the sections we examined. It may be added that in some instances the narrow lumen was occupied by an irregular mass of deep yellow pigment, everywhere in contact with the irregular inner boundary of the internal coat of the vessel. These conditions probably illustrated early stages

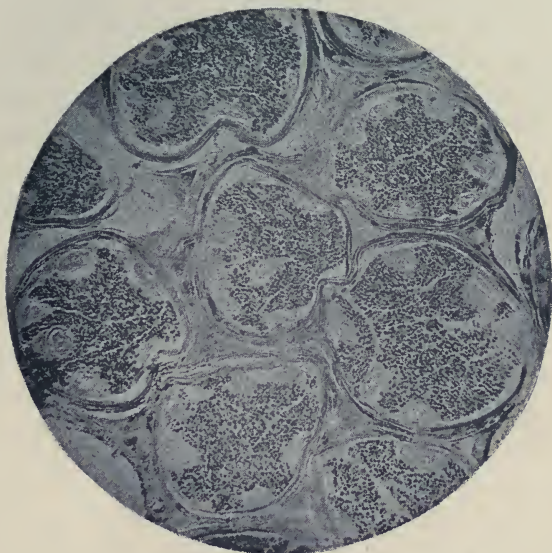


FIG. i. Transverse section of a nerve: low power. The unstained patches within the funiculi indicate plugged vessels.

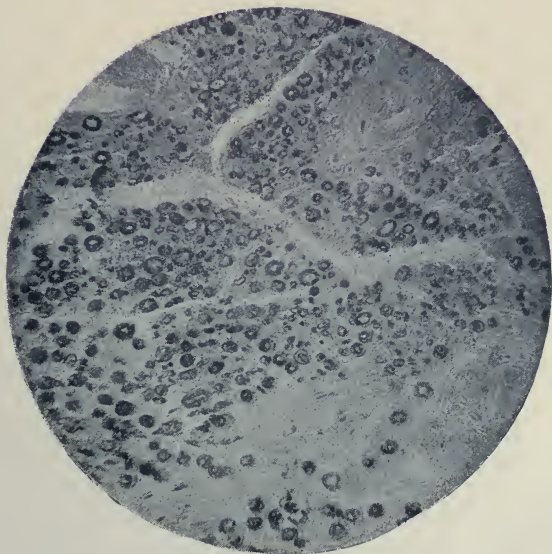


FIG. ii. Part of i. under higher power. Shows degenerated nerve-fibres. The faintly-stained areas indicate plugged vessels. (Figs. i. and ii., Weigert-Pal stain.)

in the obliterative process, as to the cause of which we are not, in the present paper, disposed to speculate.¹

Our thanks are due to Dr. C. MacPherson, Wadsley Asylum, for kindly furnishing the drawings illustrating the diseased condition of the blood-vessels.

¹ Since this paper was written we have examined certain nerves in three more cases of general paralysis. In one case the nerves at the wrist showed the changes in the vessels described above; in the other two cases no such changes were found in the nerves examined, but the experience afforded by the cases detailed above justifies us in observing that they may nevertheless have existed in other nerves not examined.

ON HEREDITARY ATAXY, WITH A SERIES OF TWENTY-ONE CASES.

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SINCE Friedreich first described Hereditary Ataxy in 1861, many cases (in all less than a hundred) and series of cases have been reported, which have been supposed to belong to this disease; but in some instances the reports have been so meagre, and in others there has been such wide variations in the symptoms present, that it has so far been well nigh impossible, from the literature of the subject, to obtain a definite idea of the conditions which must be present in any given case, in order to entitle it to a place under this heading.

The establishment of a reliable and permanent criterial picture to be used for purposes of comparison will necessarily require many years for its completion, more especially in a disease so rare as this one. The establishment of such a standard, however, worked out from a critical scrutiny of all available cases, is of the utmost importance, even if it have to undergo considerable amendment from time to time, as the stock of definite facts accumulates.

The work of Dr. P. Ladame in this direction (BRAIN, vol. xii. p. 467) is highly commendable; and to it I would refer anyone who wishes to acquaint himself with a brief historical review of the subject, as well as with the views that many distinguished neurologists have held, and still hold, regarding the disease.

After a critical review of all available published cases, and an elaborate study of a case for some time under his

own observation, which he accepts as typical, Dr. Ladame proceeds to erect a criterial standard which he summarizes as follows :

“Slow and progressive ataxy of the four limbs, usually attacking several children of the same family, dating very often from very early age—commencing in the legs, extending gradually to the trunk and arms, the muscles of the larynx, those of the tongue and eyes; weakness of the legs, increasingly difficult gait; choreiform unsteadiness; static ataxy; difficulty of articulation; nystagmus; spinal curvature; paralytic club-foot; abolition of knee-reflexes; no sensory disturbances; absence of oculo-pupillary anomalies and of lightning pains; integrity of sphincters.”

Further on Dr. Ladame states that there are no authentic cases known of onset after the twentieth year, that there is integrity of the special sense organs, and that horizontal nystagmus, static and dynamic, is rarely absent. He also speaks of scoliosis and scanned speech as common.

If the cases herein reported are accepted as instances of this disease, then the criteria as set forth in the above summary should be considerably modified. And the same may be said of the descriptions found in the text books or elsewhere, so far as my reading has extended.

CASE XVIII.—A business man, single, of temperate habits, with good family history, excepting that his mother became ataxic at about thirty-three, the disease progressing steadily, till she died of tubercular diarrhœa at forty-eight. Her mother and maternal grandmother were similarly affected, but I will not proceed farther with a verbal description of the heredity, as this may be seen by reference to the accompanying chart.

Patient was always active and vigorous in every way until attacked by his present disease, being rather among the foremost in all athletic sports and school work.

His attention was first attracted to this disease while he was working with a surveying party in Texas, and then it was noticed by others before he himself noticed it. He was then twenty years old, and the ataxia was so marked as manifested by a staggering gait, that his chief thought him intoxicated. But now he recollects that at least two years before this, or when he was eighteen years old, the draughtsmen in the office where he worked complained that he rendered their desks or drawing-boards so

unsteady when he leaned against them that they could not well go on with their work ; and he further distinctly remembers that on a certain occasion at about this time, when in company with other young men in the country, he was quite unable to read the large letters of an advertisement at a considerable distance, though each of his companions could read them with ease. He feels confident that this comparative visual defect had not always existed. I should say here that this patient has a good English education and that his intellectual capacity is distinctly above the average, so that, notwithstanding his affliction, he would at the present time be justly regarded as a well-informed man ; and in this same connection I wish to say further that I can hardly find words in which to properly express my admiration for the high humanitarian spirit and untiring energy which this gentleman has all along displayed, in assisting me to present to our profession all facts that might throw any light upon this obstinate disease.

He feels quite certain, too, and in this he is corroborated by his older relatives who have had an opportunity of observing him carefully, that at the age of fourteen, when his voice underwent the change incident to puberty, that there appeared a gradually increasing defect in utterance. His speech was slower and his syllables less definite and distinct than formerly. The ataxia has always been much more pronounced if he was fatigued, and he now remembers that at the age of eighteen he could not walk in a straight line when very much tired.

There has been some progressive loss of power in the legs from the first, but this has been insignificant throughout in comparison with the ataxia. The ataxia was first noticed in the legs and has progressed more rapidly in their muscles than in other situations, but it has been distinctly noticeable in the arms from a very early period, and has certainly progressed. An increase of the knee-jerk was probably an early symptom, because it has been so in all the other members of the series whom I have examined (in one, a younger brother of the patient, the disease is just now commencing to develop) ; and because the patient has shown me notes of his case made four years ago by such competent observers as Dr. Henry M. Lyman and Dr. Walter Hay. The former physician made, I believe, the diagnosis of ataxic paraplegia, which, at the time of his examination, and without the more complete family history since elaborated, was probably more nearly the proper nosological position of the disease than any other according to the standard text-books ; and even now I

think hereditary ataxia paraplegia is a designation that comes pretty near the mark in assigning to this series of cases a place in accordance with the usual classification of degenerative diseases of the nervous system.

At no time has there been any pain or any other disturbance of sensation. There has been no atrophy, spasm, or trophic manifestations. The sphincters have not been affected, and the patient thinks sexual power has not been more impaired than can be attributed to the general decline in bodily weight and strength which has slowly supervened within the past six years, the weight having fallen from 136 to 112 lbs. in that period.

I should have stated that a tendency to choking while eating has all along been a very troublesome symptom, and is in some way (whether mainly from inco-ordination or paralysis, I can't say) due to the tendency of food to come into contact with the larynx; and this has been a troublesome symptom in several members of the series.

For several years past vision has progressively failed, so that he could read best in a dim light.

Present Condition.—Patient is considerably emaciated, though he eats fairly and sleeps well. He is of medium size and well formed. The sensibility is normal, the knee-jerk is greatly exaggerated and equal on both sides; there is a slight ankle-clonus, the skin reflexes appear in the main normal, but the cremasteric and abdominal are not strongly pronounced. There is marked ataxia in nearly all voluntary movements; so that the patient can only walk when supported by an attendant, and can only stand when leaning against some solid support. Closure of the eyes does not materially increase his difficulty. The gait is such as would usually be described as cerebellar, the patient leans rather backwards against his attendant and sways from side to side, and he has an uncomfortable sense of insecurity all the time, as if his head must fall violently to the ground. He experiences a distinct loss of power in the legs, which he thinks is even greater than could be accounted for by his general decline in bodily vigour, but the muscles are firm and well developed. All the voluntary muscular movements are slowly performed, and of this the patient is quite conscious. He cannot reach out his hand suddenly to seize any given object. The hand moves slowly and deviates several inches in various directions from the direct line that would normally be taken in such an effort. This tardiness of movement is plainly noticed when the patient raises the eyes; on being addressed or in winking, the eyelids will rise so slowly

as to be suggestive of temporary ptosis, but in the end they are raised too high, so that the sclerotic is often so much exposed as to display an expression, usually associated with some intense emotion, when in fact the patient is suffering from no emotional disturbance whatever. Though ataxia in the muscles that move the tongue would be difficult of conclusive demonstration, because the normal movements could hardly be definitely described (and the same might be said of the muscles that move the lips), yet a careful observer would see at a glance that the movement of both tongue and lips, especially the former in this case (because a full beard is worn), were far wider in range than usual. There is no difficulty in swallowing, excepting the tendency to choking already noticed, which might be explained by ataxia and tardiness of the muscles concerned, because no extraordinary effort has to be made, and there is no tendency for liquids to pass out through the nose.

There is marked inco-ordination of the various muscles of facial expression, which is easily observed when the changes are going on incident to the discussion of an absorbing topic, the action being more marked, now in this group of muscles and now in that, so that people casually meeting the patient frequently get the impression that there is some mental defect.

There is no spontaneous movement during sleep or waking repose, but a comparatively slight voluntary movement gives rise to very extensive and peculiar movements in muscles far removed from those required for the execution of the required act. For instance, where the patient puts out his hand to take a book from a table beside which he may be sitting, the whole upper part of the body goes through a series of irregular movements highly suggestive of chorea. This is particularly the case with the head, which is somewhat inclined forward, moved from side to side, and the chin is protruded; and there is often an associated movement of the other hand. In none of these movements is there ever anything approaching a jerk.

This case and five others of the series was thoroughly examined by Dr. W. F. Montgomery, Professor of Ophthalmology in the Women's Medical College, whose reports I herewith append. And two of the less advanced cases (19 and 20) were minutely examined by Dr. Casey Wood, Professor of Ophthalmology in the Postgraduate Medical School in this city.

Patient states that for ten years past he has suffered from some gradual impairment of vision, and has had double vision at times, of not more than a few days' duration, but not for several

years past. Examination shows marked ptosis when the patient is at rest, but by an effort he can raise the lids, showing sclerotic above the cornea when the eyes are directed in a horizontal plane. There is perfect co-ordination of the ocular muscles except to extreme right, where there is slight lagging of external rectus—not enough, however, for the production of diplopia; the lids and conjunctivæ are normal.

The pupils respond to light but very slowly; and the same is true of accommodation. Dilatation also occurs slowly on stimulation of the skin of the neck.

Vision is 20/200 in either eye. Snellen No. 5 *can* be read, though with difficulty, at 8 inches in an ordinary light, much more easily in a dim light.

The ophthalmoscope shows decided blanching of the optic discs and lessening of the calibre of the arteries, with slight but distinct atrophic changes in the retina.

There are only slight peripheral limitations of the field, and almost complete colour blindness, red only being distinguished with any degree of certainty.

The above description fairly represents the patient's condition, May 2nd, 1891, when I first examined him, and there was no material change until about October 15th, except that under tonic treatment with galvanism—20 to 60 m.a.—to the spine for ten minutes two or three times a week, there was a considerable improvement in the general health with a gain of about 10 lbs. in weight. At the last-named date the patient was attacked with a severe cold, and in a day or two became unable to walk even with assistance, on account of loss of power in the legs, without any alteration of sensation or the reflexes. There was some cough, but the appetite remained good. To-day (November 13th) there is considerable improvement in the legs, but the power is much less than before this attack.

CASE XIX.—Sister of the preceding, first consulted me May 5th. She is twenty-six and has been subject to headaches with nausea and vomiting since childhood; she first menstruated at eighteen, but this function has been too infrequent since, often occurring at intervals of from two to four months. Ataxia first showed itself in the legs when she was twenty, but had not invaded the arms, the organs of speech or the ocular muscles to any considerable degree till she was twenty-three. She then began to notice that vision was indistinct in a bright light, and she would move back from the window when reading. The ataxia had advanced so far in the legs that she was obliged to give up dancing.

Present Condition.—Patient is well formed, and of medium size; she has intellectual faculties above the average and a happy and cheerful disposition. She is in a rather reduced state of general health, as manifested by numerous very painful boils about the hips and thighs, which have been appearing in successive crops for the last two or three months.

The cutaneous and muscular reflexes are all distinctly exaggerated, especially the knee-jerk.

There is not and has not been any disturbance of sensation or of the sphincters. She walks well without assistance of an even surface, but deviates considerably from a straight line, no matter how hard she attempts to prevent it. Closure of the eyes does not materially increase the tendency to fall. The same ataxy of the movements of the hands, eyelids and face are present, as in the preceding case, and the same tendency to extensive associated movements, alike in quality but less in degree. There has been no tendency to choking.

The oculists report slight static ptosis with inco-ordinate overaction of the levators. All the movements of the ocular muscles and all the pupillary reflexes are sluggish. All are present, however, and in no case can nystagmus be produced. Vision is 20/80 and Snellen No. 3 is read well enough at 10 inches. Patient reads much better in a dim light. The average expert would pronounce the fundus normal without knowing anything of the condition of vision.

The colour sense is not much affected and there is some peripheral limitation of the field of vision.

The patient states that she is conscious of some difficulty of articulation, but there is no markedly positive defect.

It should be stated that this patient, the only girl in a family of five children, was, before the onset of this disease, very active physically and accustomed to join in the sports and exercises practised by her brothers; and indeed, all the members of this family were very active, so that in athletic contests at school they very frequently carried off the prizes. For this reason it is easier to determine the date of onset of ataxic symptoms than it might be under other circumstances. With the application of the galvanic current twice a week, the occasional exhibition of fractional grain doses of calomel, six week courses of Fowler's solution carried up to six drop doses after meals, and several short courses of simple bitters with port wine, the boils, the headaches and the irregular and painful menstruation have entirely disappeared, and the special symptoms of the disease have certainly not increased.

CASE XX.—Brother of the two former, æt. twenty-four, well developed, cheerful with correct habits, good business man. The disease began, without exciting cause, at age of eighteen. The unsteadiness of gait and difficulty of articulation came on together, and not until two years later was a defect of vision realized. He has carried a cane for the last few months, but can walk fairly well without one, though he deviates considerably from a straight line in walking. The defect in speech is marked, the labials are often defective; yet he whistles without much difficulty. Occasionally the labials are exploded.

The ocular condition is essentially identical with that of the preceding case according to the oculist's report and my own observation, excepting that ptosis is considerably more marked in the left than the right eye.

There appears to be a general relaxation of the facial muscles in repose and even during commonplace conversation, giving the patient a heavy and stolid expression which is quite at variance with the prevailing state of his mind.

He has had galvanism to the spine, as No. 18, and thinks there has been distinct failure in the power of walking in the past three months.

CASE XXI.—There are two other children in the family, twins æt. twenty-one, in one of whom there is distinct exaggeration of the cutaneous reflexes and slowness of speech, but no other indication of the approach of the disease. The remaining twin is entirely free, up to the present time.

In the next family three cases have occurred.

CASE IX.—Is a farmer's wife, aged sixty-seven, of regular habits, who has borne three children. The disease first showed itself at the age of about thirty-five, by ataxy in the gait, and in the hands a year or two later. At the age of about fifty she could walk only with assistance, and at fifty-eight could not pour tea. For several years, at about this time, she went about the house on hands and knees, but for the past four years has been entirely helpless.

Failure in articulation and vision in right eye began to be manifested at about fifty-seven and advanced very rapidly. Vision with the eye first affected was nearly gone in three years, when it began to fail in the other, about the same course being run. No defect of sensation or the sphincters. Memory has failed much within the past four or five years.

Present Condition.—Permanent spastic contraction of the legs, the three being flexed at about a right angle, which can be partly

overcome by force slowly and steadily applied. When the patient attempts to talk the tongue appears to move in every conceivable position without being protruded; the face undergoes various inco-ordinate movements, the head is repeatedly bent forward and moved from side to side, there is protrusion of the chin, and the arms were flexed and moved forward and backward. The movements are highly suggestive of chorea but less rapid. In articulation there is a marked dwelling on the vowel sounds. When I briefly explained the nature of my errand, the patient partly succeeded in saying she was glad to see me, said she had no pain when I forcibly extended her legs, and correctly identified the parts touched when sensibility was being tested. But she said she had only been blind about two months, showing her great failure of memory.

Oculist's Report.—Husband states that patient used to be a great reader and was able to read good print till about nine years ago. At present she can only just distinguish a bright light.

There is almost complete ptosis. If the lids are held open, as is necessary in making an ophthalmoscopic examination, they remain open for a short time and then slowly close. There is scarcely any action of the external ocular muscles, the eyes staring directly forward when the lids are raised. Pupils of medium size and not responsive to light; media clear. The discs present a typical picture of complete atrophy of the optic nerve.

CASE X.—Son of No. 9; farmer, age thirty-eight, single, temperate. First noticed disease in legs at age of thirty-one, and in his hands about two years later. Defects in vision and articulation were first noticed at the age of thirty-five, at which time he began to use a cane. Has occasionally had shooting pains in the legs for the past two years, not very severe, however; for the past few months has not been able to retain his urine as usual, and has had to urinate much more frequently than before, having to make great haste to prevent an accident.

Excepting that he still walks with a cane, and that there is more exaggeration of the reflexes, there being well marked foot-clonus on both sides, the objective symptoms in this case are identical with those already described at length as belonging to Case No. 18. There is no alteration in sensations, as might be suggested by the pain and the bladder symptoms. The oculist's report is also practically identical with that of Case No. 18. The general health of this patient was good, though he said he had lost a few pounds in weight.

CASE XI.—Brother of No. 10; died of exhaustion of this disease at age of twenty-six. First symptoms appeared at age of sixteen, as simultaneous ataxy of the legs and eye muscles. He walked with a cane at nineteen, and was helpless at twenty-one. Voice and vision were both somewhat affected at seventeen, but not very markedly so at the time of his death. There was no defect of sensation nor of sphincter action.

Though I had already obtained a pretty full history of the next three cases from their near relatives, I happened to learn that so competent an observer as Dr. Norman Bridge of this city was temporarily living very near them (about 2,000 miles from here), and he examined them and kindly sent me the following notes:

CASE V.—“Patient is sixty-seven years of age. She has a shrunk, cadaveric body, her muscles having undergone extensive atrophic change; the head is in almost constant motion, much like that produced by chorea; this is especially the case when she is talking, moving her hands, or conscious of being observed; the only difference is that the head movements are less regular than in localized chorea. When lying or sitting she can move her legs and even kick, but these voluntary movements are performed with some ataxic irregularity. She is utterly unable to rise from a chair, and has been so for the past year. She moves her hands and arms about freely, but with the shaking, irregular movements peculiar to the ataxic. She cannot, with her eyes closed, accurately touch her nose nor any other desired point on her body. The voice has the same irregularity as the hand movements; it is not a tremor, but a shaking; she does not hesitate in her enunciation, but, so to say, shakes her words out with poor but not abolished labial and palatal action.

“There is no contraction anywhere. The knee-jerk is considerably exaggerated, but there is no foot-clonus. The reflexes are alike on each side. Nystagmus is absent, but the eyesight is so much impaired that she can only see the spots on playing cards (she plays solitaire much of her time). I tried to make an ophthalmoscopic examination, but under great difficulties, so that I do not feel able to say with any degree of positiveness what the condition of the fundus is. Vision is the same in each eye.

“There is no constipation nor vesical symptoms of any sort. Attempts to swallow fluids often provoke coughing and strangling, doubtless from their entrance into the trachea to some extent. She has learned to swallow with great care and deliberation. Her appetite is poor, tongue clean; she insists that her soft

palate is too low, and it is, in fact, relaxed; she insists, also, that she has intense heat in her back, but to my touch it is normal. Pulse 112. The pupils are only very slightly responsive to light."

I have given Dr. Bridge's notes in detail because I thought it important to have an entirely independent statement from so high a source. I will add that the first symptoms in this case appeared, at thirty-five, in the legs, and has progressed quite steadily down to the present condition. There has been no pain or sensory disturbance, unless the intense heat in the back be so regarded.

CASE XV.—Son of No. 5; aged forty-nine, also examined by Dr. Norman Bridge, whose notes I quote: "He walks with an ataxic gait, and his upper extremities show to all tests the same condition as the lower. Patient states that the disease first appeared in the lower extremities four years ago, and in the upper a year or two later; says that the uncertainty of gait is much greater sometimes than at others; walks with a cane, and sometimes has great difficulty in rising from his seat. The knee-jerk is enormously exaggerated, and there is a typical ankle-clonus, these conditions being alike on both sides. There is no nystagmus, no contractions, and no bowel nor bladder symptoms.

"The sight of the left eye is abolished, and has been so for the last four or five years. It has slowly declined since he was serving in the army at the age of about twenty. The sight of the right eye is much impaired, but he can read with a good lens, and says that the print then looks correct to him. Careless swallowing of fluids is apt to carry some into trachea and set up coughing. His voice is like that of his mother's, only in a less degree; he says that his voice was normal until a year ago. He has lost 22 lbs. in the last three or four years. Pulse (sitting) is 104. His face has a congenital twist, it is noticeably longer on the left side. He is apathetic. He denies ever having had any pains in the back or extremities."

I will add that there have been no sensory symptoms, and that acute observers among his relatives have assured me that positive signs of ataxia in the gait were present at forty.

CASE XVII.—Female, sister of the preceding, was first affected at fourteen and died from exhaustion, extremely emaciated, at twenty-eight. The disease developed rapidly in her case, and early in its course she had severe pains in the hips and legs, and marked paralytic constipation. The voice and vision were much involved.

CASE XVI.—Male, aged forty-two, son of No. 5, brother of 15 & 17, has shown some ataxy for the past two or three years; quite obtrusive when tired, but not otherwise. Ataxia of gait the only symptom.

CASE VI.—Female, aged sixty-three. First symptoms at forty-five, consisting of ataxy of legs. The course of the disease has been slow. She can still walk on an even surface without assistance, but has to go very slowly and sways about considerably, usually steadying herself by holding on to furniture. The hands are certainly involved, but not greatly, as she can still do needlework and pour tea. Vision is considerably affected, but she can read coarse print in a dim light. Cannot read in a bright light. Speech has become very slow, but is quite distinct. General health fairly good. Other symptoms negative. History given by a relative, himself affected, who has seen her almost daily for years.

CASE VII.—Female, died at forty-six of tubercular diarrhœa. First affected, at twenty-eight, with slight ataxy of hands; a few years later it was noticed in the legs, but it progressed very slowly till she was thirty-five when, after child-birth, it advanced more rapidly. With the exception of the last six months, when she became greatly emaciated from chronic diarrhœa, she could walk alone, but would deviate several feet to either side of her line of direction. Until the last two years of life was able to pour tea. Her voice gradually became very slow and somewhat indistinct, but this did not show itself till about the fortieth year and progressed very slowly.

Vision began to be impaired at thirty-six, and by the time she was forty she could only read very large print in dim light; after forty vision failed more rapidly. Other symptoms negative.

CASE VIII.—Female, died at thirty-two, of consumption, only slightly affected. Two of her children are living, aged respectively twenty-two and twenty-five, and are healthy.

CASE II.—Female, died at about sixty-five. The disease was well developed in all its usual features at fifty, and for the last fifteen years of life the gait showed cerebellar divergence, if I may use that term, to almost the same extent as obtained in No. 7. This statement is the result of a conference among her surviving children and other near relatives, and though I did not attempt to extract from them evidence of earlier symptoms, it is pretty safe to say, judging from the other cases, that they existed at forty.

CASE IV.—Male, died at about forty; I could not learn the

cause of death. He was only slightly affected for two or three years preceding death. No further particulars available.

CASE XII.—Female, died of pneumonia at thirty-one. Married at twenty-three and had four children. First symptoms appeared at about twenty, consisting of ataxy in the hands and arms, three or four years later the legs became affected. The disease progressed quite rapidly in her case. Was unable to walk without assistance at the age of twenty-eight. No pain at any time. Mind impaired during the last two or three years of life—hebetude mainly.

CASE XIII.—Female, aged twenty. First symptoms noticed consisted of ataxy in legs, beginning at fourteen. The arms were involved in a few months, and vision and voice failed rapidly. The voice is described as slow, harsh, and indistinct. There have been darting pains in the legs, but she still walks with a cane. This and the preceding patient live 1000 miles away, and the above incomplete history was obtained by correspondence carried on by a near relative. I hope later to get a more exact report.

The same correspondent informs me that he has learned from a reliable source that No. 12 has a younger child distinctly affected, but has no further particulars.

CASE III.—Male, died at fifty-five of pneumonia. First affected at forty, after a fall; was able to walk with a cane at time of death. Arms, voice and vision distinctly, but only slightly, affected.

CASE XIV.—Male; died of phthisis at sixteen; affected at eleven; the symptoms began in legs after a fall; he was never able to walk afterwards. Later they appeared in the arms. I have no information as to voice and vision.

CASE I.—Female, died at seventy-two. The first symptoms appeared at forty-five, after the patient had sustained a fracture of the hip by a fall. They consisted in marked ataxia in the hands; after injury she was never able to walk without assistance. No more definite or satisfactory account of the symptoms can be obtained. Various relatives who remember her well, and have since seen numerous and well marked instances of this disease, are certain that she was affected with it for many years prior to her death, in the same way as the other advanced cases.

CASE XXII.—A girl of eight has ataxia of the legs with spastic symptoms so that she walks on tip-toe. Her symptoms have developed gradually during the last two or three years. I have not seen the case, but my information is reliable, though meagre.

CASE XXIII.—A boy of six, with paralysis of both legs, from anterior polio-myelitis (?), at the age of two.

CASE XXIV.—A boy of thirteen has never learned to walk, though the legs are not stiff or atrophied. He is reported as all right in every other way.

This completes the list so far as my present information extends.

Cases XXIII. and XXIV. are only cited as having a collateral bearing on the subject; and though there is some probability that XXII. and XXIV. are affected with this disease, I could not reasonably insist upon it from my present information.

In Cases I. and IV. the history is meagre, and though the period at which patient No. 1 lived is remote, yet the fact that this series of cases has been confined to families who have always maintained a good position in society, and whose family record has been well kept and can be readily traced, and also the very obtrusive nature of the symptoms, together with the fact that they are in most cases progressive through a variable number of years, warrant a diagnosis of a comparatively meagre history. For my own part I have become intimately acquainted with several branches of the family, and particularly with that branch from which I have had most of my information; and I can vouch for their superior integrity and intelligence.

Four cases—Nos. 18, 19, 20 and 21—have been under my observation steadily for seven months, and Nos. 9 and 10 for three months; and two cases, Nos. 5 and 15, have been well examined by Dr. Norman Bridge.

Taking these cases alone for a text and assuming them to be cases of hereditary ataxy, the following diagnostic criteria might fairly be deduced:

Hereditary ataxy is a disease which may be traced through several—at least four—generations, increasing in extent and intensity as it descends, tending to occur earlier in life and advance more rapidly. It usually attacks several members of the same family. It occurs most frequently between the ages of sixteen and thirty-five, but it may begin as early as eleven and as late as forty-five. It shows no

marked preference for sex, but it descends through females four times as frequently as through males. Atavism rarely occurs. The influence of an exciting cause can rarely be demonstrated, but in some instances a fall or injury has appeared to determine the onset; and any cause like child-bearing or lactation, which very much depresses the vital forces, may produce a rapid advance of all the symptoms. There is always considerable inco-ordination of all the voluntary muscles, and a sluggishness of the movements which they produce when the disease is well established. This is usually noticed first in the muscles of the legs, but in a few months or years extends to the arms, face, eyes, head, and organs of speech. Sometimes it occurs first in the upper extremities, and sometimes in the organs of speech.

The ataxy is often extreme, and the gait devious, the patient deviating several feet on either side of the intended line of progression, before he loses the power of walking. The ataxy is not markedly increased by closure of the eyes. The sense of posture is perfect.

Some weakness of the muscles of the legs, without atrophy, is frequently an advanced symptom, and occasionally there is permanent spastic contractions of the legs. In developed cases there are usually extensive choreiform movements of the head and often of the arms accompanying all voluntary movements. These irregular movements occur in the hands, legs or head whenever it is attempted to maintain either of these parts in a fixed position by a voluntary muscular effort. Movement ceases during sleep. The pulse rate may be increased to 112 in advanced cases or may be normal.¹

There is usually some degree of static ptosis, with overaction of the levator on looking upward. In rare cases there may be temporary diplopia, in the early stages, due to weakness of the external rectus. There is no nystagmus of any kind.

Atrophy of the optic nerve is a constant and early symptom, and usually progresses slowly with the other

¹ I am inclined to attribute the pulse rate noted by Dr. Bridge to emotional disturbance, because I have made repeated examinations in the six cases under my care and found it only subject to normal variations.

symptoms. Rarely it begins earlier in one eye than the other.

The response of the iris to light and accommodation is sluggish and diminishes with the advance of optic nerve atrophy; when this latter is complete, as may happen in advanced cases, there may be complete internal and external ophthalmoplegia.

There is always marked disturbance of the articulation, probably due to inco-ordination of the muscles concerned, for weakness cannot be demonstrated. In some cases there is a troublesome tendency to strangulation in swallowing liquids, due to their getting into the larynx, but otherwise swallowing is in no way difficult.

Occasionally the sphincters are slightly, but positively, affected, this symptom only appearing in those cases where spontaneous pains in the legs co-existed, having some of the characteristics of those occasioned in locomotor ataxy. Excepting the spontaneous pains already mentioned, there is no disturbance of sensibility. There are no vaso-motor or trophic symptoms, but there is a marked tendency to emaciation; there is no hypertrophy or valvular lesions of the heart.

The knee-jerk is always exaggerated and there is frequently ankle-clonus, and the cutaneous reflexes are also always exaggerated, but to a less degree. The exaggeration of the reflexes is an early symptom, and they often decline considerably when the disease is far advanced.

There is never paralytic club-foot, nor any other deformity excepting rarely permanent spastic contractions of the legs in advanced cases. In none of these cases have the patients ever suffered from rheumatism so far as I can learn.

I wish to repeat that the above summary of the symptomatology of hereditary ataxy is only intended to apply to this particular series of cases; and I have only presented it in this way so that it might be the more easily compared with other series.

I regret that I am unable to show any sections illustrative of the pathological anatomy of the disease. Of the pathology it seems pretty evident that the prominent fea-

tures consist in an extensive degenerative process affecting mainly in the upper motor segment, that is the cortical cells and the fibres extending from them to the cells in the cord and medulla ; and that the tissues concerned have derived a deficient vital endowment from the parent ; this deficiency manifesting itself as frequently after, as during, the age of development.

The optic nerve atrophy, the occasional spontaneous pains, associated with muscular weakness of the bladder and rectum, together with a tendency to emaciation, suggest that tissues outside of the upper motor segment may be either primarily or secondarily involved.

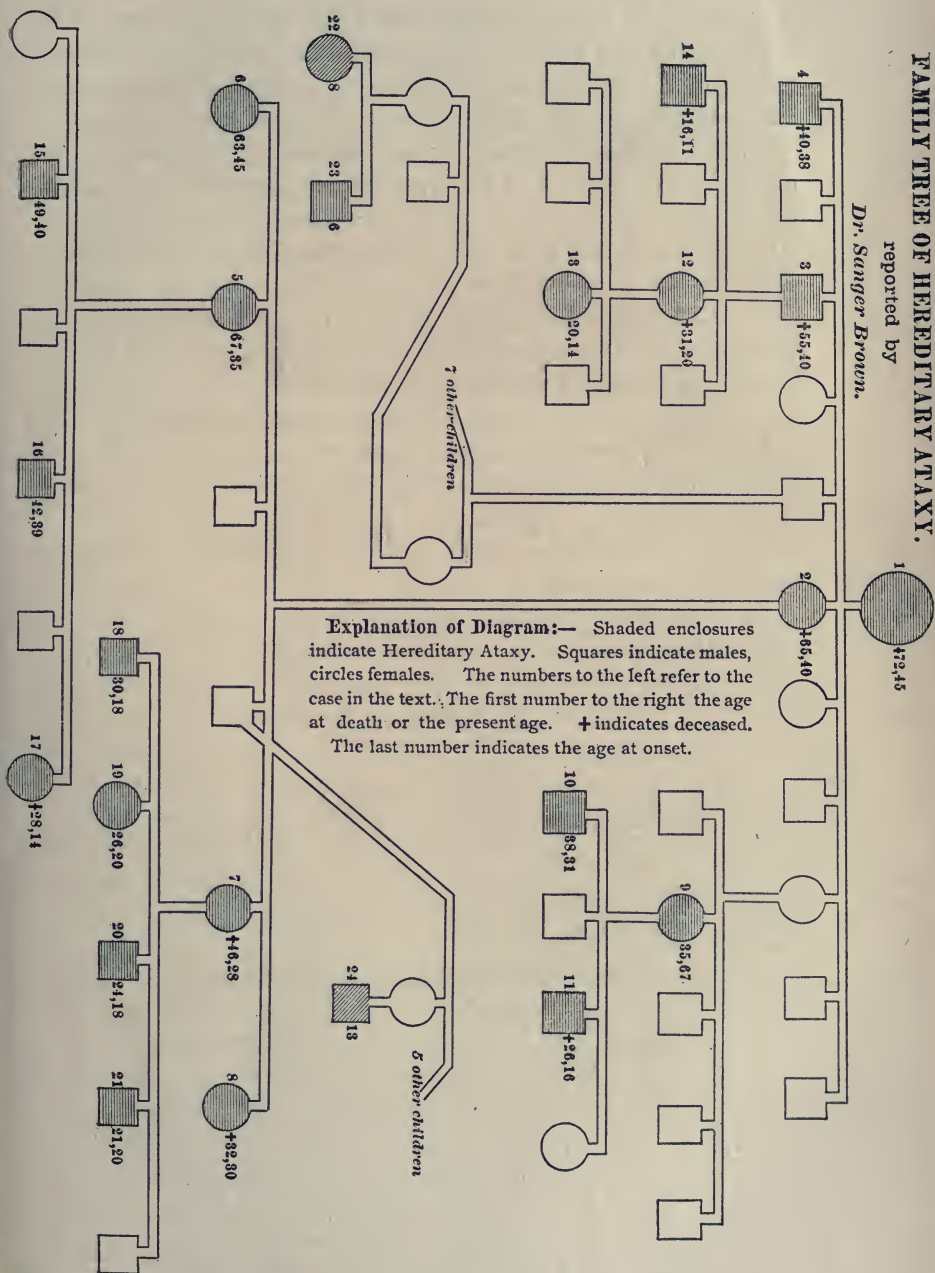
The integrity of the muscular sense and the peculiar nature, wide extent and extreme degree of the ataxy, together with the state of the reflexes, suggest that the difficulty lies rather in the efferent than in the afferent paths.

The degeneration is uniformly progressive, though it presents considerable variation in the rate at which it advances in different individuals and at different times in the same individual. I have applied galvanism along the spine in three cases about three times a week for the last five months, using a current varying from 25 to 60 m.a. for ten minutes. The more advanced patient has an impression that the strong current did harm, and he is inclined to attribute the recent considerable loss of power in the legs to that cause ; and while such an influence cannot be certainly demonstrated, I should in future cases hesitate to apply a current of more than from 20 to 30 m.a. I changed the poles usually several times during each sitting.

I have paid careful attention to the general health, using wine with a bitter tonic and arsenic from time to time ; and all the patients have gained several pounds in body weight. In the case least affected, I cannot observe any advance of the symptoms, but in one there has been an advance of the ataxy noticeable in the gait ; and in the third a somewhat rapid onset of weakness in the legs coincident with a bad cold, already mentioned. Now, after about ten weeks, muscular power in the legs has returned to a considerable degree, but not so as to permit the patient to walk.

FAMILY TREE OF HEREDITARY ATAXY.

reported by

Dr. Sanger Brown.

The main points of difference between the symptoms observed in my series, and the criteria established by Dr. Ladame, I will not occupy space to further indicate, as I have already gone far beyond my intended limits.

With reference to the etiology and symptomatology set down by Dr. Gowers in his text-book, if the age at the time of onset were considerably advanced, and if it were stated that the knee-jerk and skin reflexes might be greatly exaggerated, that in some cases there might be ankle-clonus, and that there might be optic atrophy, the cases herein reported would be fairly well provided for. Indeed, it would seem that there may be much more variation in the age at which the disease first makes its appearance, the distribution and intensity of the pathological process and the consequent symptoms, than has hitherto been suspected.

REMARKS ON DR. SANGER BROWN'S CASES.

I.—BY DR. ORMEROD (LONDON).

Dr. Sanger Brown brings forward a long series of interesting cases, broadly characterised by the title of hereditary ataxy. Ataxia, indeed, was the leading symptom, while the hereditary nature of the disease in the families of this stock is sufficiently obvious. As to the date of onset¹ Dr.

¹ The dates of onset, given in his diagram, fall as follows:—

1st Generation—

Case 1, F, at 45; died at 72

2nd Generation—

Case 4, M, „ 38; „ 40
 „ 3, M, „ 40; „ 55
 „ 2, F, „ 40; „ 65

3rd Generation—

Case 14, M, „ 11; „ 16
 „ 12, F, „ 20; „ 31
 „ 6, F, „ 45;
 „ 5, F, „ 35;
 „ 7, F, „ 28; died at 46
 „ 8, F, „ 30; „ 32
 „ 9, F, „ 35;

4th Generation—

Case 13, F, at 14;
 „ 22, F, doubtful
 „ 23, M, „
 „ 15, M, at 40;
 „ 16, M, „ 39;
 „ 17, F, „ 14; died at 28
 „ 18, M, „ 18
 „ 19, F, „ 20
 „ 20, M, „ 18
 „ 21, M, „ 20
 „ 10, M, „ 31;
 „ 11, M, „ 16; died at 26

Sanger Brown says, "it occurs most frequently between the ages of 16 and 35, but it may begin as early as 11 and as late as 45;" as it descends through the family it tends to occur earlier.

As a rule no cause can be assigned for it; but any debilitating influence hurries its progress. This progress is slow in most cases. In two cases, which appear to have been rapid, death took place by exhaustion from the disease in 10 and in 14 years.

The principal symptoms are ataxia of the limbs, usually beginning in the legs and progressing upwards, rendering the patient in time helpless, though muscular power is retained (enfeebled in some instances). With this is associated excess of tendon reactions, often ankle-clonus and sometimes contracture. The skin-reflexes are also exaggerated. No sensory symptoms, except pains in some exceptional cases. As the disease progresses, "extensive choreiform movements of the head, and often of the arms, accompanying all voluntary movements," are seen. "Then irregular movements occur in the hands, legs, or head, whenever it is attempted to maintain either of their parts in a fixed position by voluntary effort."

Articulation is interfered with, apparently by similar kinds of uncontrollable associated muscular action. Sometimes there is a tendency to choke when swallowing. The eye symptoms are peculiar; thus there is a drooping of the eyelids, resembling ptosis, but attempts to raise the eyes may result in overaction of the levators of the lids, giving a staring expression. In one case there appears to have been actual ophthalmoplegia externa. Vision fails, often at an early stage, from a progressive optic atrophy; the pupillary reaction to light is defective, probably on account of this optic atrophy.

Thus it will be seen that Dr. Sanger Brown has described a novel and extremely interesting type of hereditary nervous disease. He himself is inclined to assimilate it to the type known as Friedreich's disease, to which the name of "hereditary ataxia" has hitherto been solely assigned. He holds apparently that the clinical picture of Friedreich's disease

(for which he adopts the excellent description given by Dr. Ladame, in *BRAIN* (vol. xiii. p. 467) as usually given, should be so modified as to include his new class of cases. To this suggestion we cannot, as yet, assent. Let us first limit ourselves, as the author does, to the discussion of clinical facts. The resemblances between the two types of disease are these:—That they both run in families, and both present spinal symptoms, the main symptom indeed being common to both, viz., ataxia beginning in the legs and progressing upwards, and causing when it first affects the legs an unsteady swerving “cerebellar” gait, rather than the jactitation of the legs which is ascribed to true tabes. With this there is an absence of sensory symptoms, at least in the early stages of both diseases. Such are the main resemblances. Others may perhaps be adduced; first the affection of speech, secondly the irregular “choreiform” movements. It is difficult to pronounce upon either of these symptoms without personally investigating cases; but we suspect from the description (1) that the movements are of an exaggerated character, in comparison with the twitchings and unsteadiness usually witnessed in Friedreich’s disease, (2) that the explosive speech differs from the drawling imperfect articulation of Friedreich’s disease. Then come in the differences, which seem really to make the clinical pictures very unlike. Thus in Dr. Sanger Brown’s cases the tendon-reactions are exaggerated, even from an early stage, and contracture may set in, suggesting, as he rightly says, a primary disease of the lateral columns; whereas in the vast majority of cases of Friedreich’s disease the tendon-reaction disappears early, and contracture, though it may occur, is a subsidiary symptom, for (as morbid anatomy has shown) the posterior sclerosis is always more marked and more advanced than the lateral. Those deformities which do commonly occur in Friedreich’s disease, viz., lateral curvature of the spine and club-foot, have not been found by Dr. Sanger Brown. The eye symptoms are quite different; thus nystagmus occurs in Friedreich’s disease, not in Dr. Sanger Brown’s type; and the peculiar action of the levatores palpebrarum which the latter author describes, the ophthalmoplegia and the optic atrophy, are unknown in Friedreich’s disease.

On clinical grounds therefore, while we admit the existence of a novel and interesting type of hereditary nervous disease, bearing very important relations to Friedreich's disease, we do not feel justified in identifying the two types. Such identification cannot in fact be made without an appeal to morbid anatomy. Now we know something about the morbid anatomy of the older type, viz., that the nervous centres are found to be smaller than normal, and that there is degeneration of the posterior columns, which in all the *post-mortems* yet made has been of an advanced character, coupled with a less advanced degeneration of the pyramidal tract, and possibly with some other lesions. But Dr. Sanger Brown has not yet had an opportunity of examining any of his cases *post-mortem*.

We will, therefore, collate another set of cases, which appear to bear a close resemblance to Dr. Sanger Brown's; and in one of which a *post-mortem* and elaborate microscopical examination was made. These are published by Dr. M. Nonne, in the *Archiv. für Psychiatrie*, vol. xxii., 1891, p. 283. The accounts relate to three brothers, by name Stübe.

CASE I.—Heinrich Stübe, æt. 46 at time of examination. (See Genealogical Table, p. 277.)

Was a sailor for fifteen years; was shipwrecked when about 27 years of age, nearly drowned, and exposed for three days and nights. Shortly, but not immediately afterwards, a weakness and uncertainty in the legs began, which progressed, and in two years' time extended to the hands; his speech also becoming affected. In another two years he became practically demented and refractory.

When seen by Dr. Nonne his head was small, forehead low, occiput flat. He walked with crutches (and indeed could hardly stand without them), gait straddling, eyes fixed on ground. Swinging of body when he tries to stand without crutches, not increased by closure of the eyes.

Eyes.—Normal reaction of pupils. Difficulty in ocular movements; downward movements normal, the movements outward, inward, and of convergence, are limited, up-

ward movements still more so. Nystagmus both during movement and during fixation. Optic atrophy moderately advanced, with limitation of field of vision, and impairment of central vision (V. ; L. = $\frac{5}{10}$).

Facial movements are performed with unnecessary expenditure of energy, and with them are associated abnormal and extraneous movements, *e.g.*, wrinkling of the forehead and eyebrows when he whistles.

Speech immoderately loud, nasal ; the words are blurted out.

Upper Limbs.—Slight inco-ordination in fine movements, *e.g.*, in writing. Muscular power normal ; sensibility normal in all forms. Tendon reactions normal. No rigidity when passive movements are executed, but after voluntary movements he appears to have some difficulty in relaxing his muscles.

Lower Limbs.—Inco-ordination is manifest when he attempts a given movement, but no loss of muscular power. Knee-jerk lively, but not abnormal ; no ankle-clonus. Tendency to “active tension” of muscles as in upper limb.

Mentally, some weakness ; thus, he would accuse his brother of cheating and robbing him.

The condition had been stationary during the last few years.

CASE II.—Fritz Stübe, æt. 49 at time of observation.

At 14, noticed an awkwardness of the legs, particularly in going up ladders, walking over planks, &c., and then progressive weakness of the legs, so that at 21 crutches were necessary. Some years later weakness and uncertainty in upper limbs. After some years more, an affection of speech, which rather rapidly reached its present stage. No cause could be assigned for the disease. The symptoms had been stationary for the last ten years.

A somewhat slight and bowed man ; head (especially occiput) small, forehead low ; mental faculties below par. Stands even more unsteadily than Case I. ; unsteadiness not increased by closing the eyes. Manages to walk in a straddling way, by pushing a go-cart in front of him ; falls forward without this support.

Eyes.—Some uncertainty in fixing; nystagmus during movement; outward and upward movements limited. Pupils slightly unequal, react to light. Optic discs sharp and pale; field narrowed concentrically to $\frac{1}{2}$ normal; central vision impaired (V.; R.= $\frac{1}{8}$, L.= $\frac{1}{9}$).

Facial movements exaggerated; wrinkling of forehead, &c., during speech.

Speech loud, nasal, explosive, the breath appearing to be badly regulated (the movements of the uvula, soft palate, &c., appeared in both cases, I. and II. to be normal.)

Upper Limbs.—Inco-ordination in such movements as buttoning, picking up small objects, &c., more marked than in Case I. *Lower Limbs.*—Patellar tendon-reactions lively but normal; skin reflexes feeble; muscular power and sensation normal in all limbs; some prolongation of muscular contraction as in Case I.

CASE III.—August Stübe, æt 40.

From the age of 10, his friends had noticed awkwardness and uncertainty of movement; abnormality of speech as long as he can remember: his tongue “seemed too short;” difficulty in learning.

A thin anæmic man, with slight kyphosis, and signs of chronic pulmonary tuberculosis; mental enfeeblement; head small; low forehead; flat occiput.

Speech (as in last cases) nasal, and showing defective control over the muscles employed; the inspiration and expiration being particularly badly regulated.

Eyes.—Pupils equal, reacting rather slowly; eyes unsteady during fixation and showing nystagmus on movement; upward and outward movements even more limited than in the other cases. No ophthalmic examination was made.

Can only stand with his back against a wall; cannot sit down or rise from sitting without help; can only walk when supported on both sides. When he thus walks, there is no stamping or jactitations of the legs, but a certain unregulated innervation goes on, which calls into action other muscles than those required for the act.

In the upper limbs there was inco-ordination of movement, particularly well seen in the attempt to write.

Tendon reactions feeble in upper limbs ; but kneejerk lively. Sensation normal. Difficulty in relaxing muscles after voluntary contraction, but no true contracture.

Evidently, then, these three brothers were affected with the same disease ; it attacked the two last mentioned brothers at the age of 14 and 10 respectively, and the other not until he was nearly 30. It ran a slowly progressive course, finally settling down into a stationary condition. The symptoms (to recapitulate briefly) were as follows : progressive ataxia first of the lower then of the upper limbs, without gross loss of muscular power, without contracture (though a peculiar difficulty in relaxing the muscle after voluntary action is mentioned), without affection of the sphinctus, without affection of sensation, without material alteration of tendon or skin reflex ; peculiar nasal explosive speech, suggesting defective regulation of the vocal cords and respiratory muscles, and accompanied (in the first two cases) by facial movements, over action of the facial muscles during movements of expression, nystagmus, limitation of ocular movements especially in the upward and downward directions, optic atrophy (in two cases). Mental feebleness.

The resemblances between Dr. Sanger Brown's cases and these of Dr. Nonne appear to us very strong indeed, the only differences being that in the second set there were no symptoms of lateral sclerosis (exaggeration of tendon-reaction and contracture) ; and that there was nystagmus, with perhaps more definite paresis of ocular movement and less of the peculiar over-action of the levatores palpebrarum. Mental degeneration also appears to have been more prominent.

If then we may infer from the symptoms that the disease was in both instances the same, we shall turn with all the more interest to the morbid anatomy of Dr. Nonne's third case. This patient (Case III., August Stübe) died of pneumonia which complicated his previously existing phthisis. The anatomical changes were far less extensive than might have been expected from the symptoms ; and may be very shortly summarised under two heads.

(1) Smallness of the nerve-centres—cerebrum, cerebellum, pons, medulla, and cord. (For the exact measurements we must refer to the original paper). This atrophy was unaccompanied by any sign of inflammatory change; and is therefore considered by the author to be primary, and dependent on defective development.

(2) A disproportion in the relative numbers of large and fine nerve-fibres in the spinal nerve-roots. The fine nerve-fibres were greatly in excess (? atrophy or non-development of the large fibres). This anomaly was most marked in the lumbar nerve-roots, and more marked in the anterior than in the posterior nerve-roots.

No degeneration of any spinal tracts was found; indeed the atrophy of the optic nerves (in cases 1 and 2) appeared to constitute the only indication of positive degenerative disease. This therefore constitutes a salient point of difference from Friedreich's disease.

The author does not attempt to explain the symptoms on the basis of the *post-mortem* data; but he remarks that they most nearly resemble those of cerebellar atrophy. The smallness of the nerve-centres is a feature also in Friedreich's disease: indeed Schultze has suggested that it is the primary factor, and that such congenital poverty of development prepares the way for that degeneration of the spinal nerve tracts which has hitherto always been found. (*Post-mortems* upon *early* cases of Friedreich's disease, and upon members of a diseased family who have not yet shewn definite symptoms are much to be desired).

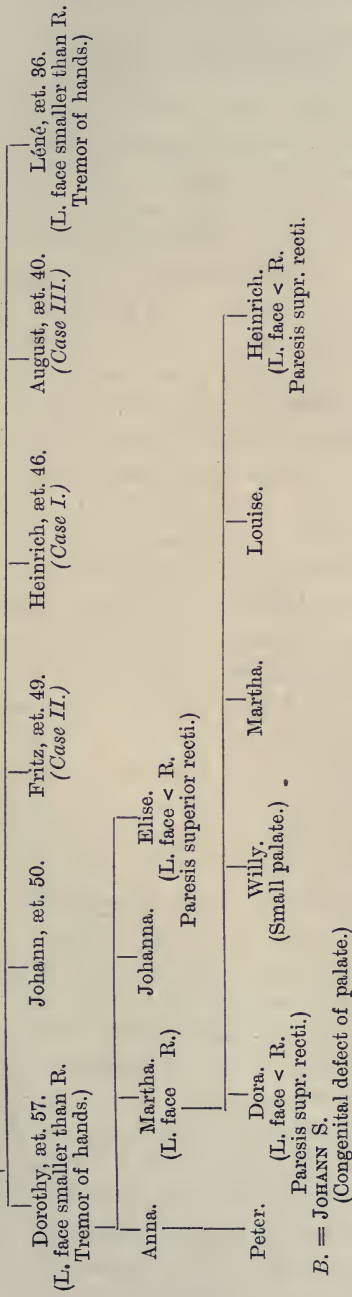
One more case must be briefly abstracted, because it exhibited during life symptoms like those we have already quoted, and *post-mortem* cerebellar atrophy in conjunction with systematic spinal degeneration, resembling that of Friedreich's disease. The account is by Dr. Menzel, *Archiv. für Psychiatrie*, &c., vol. xxii. (1891). The patient had the following family history (we notice only the diseased members). Mother healthy when young; later, developed shaking of the head, and an unsteady gait. Of six brothers and sisters, one brother became affected whilst serving as soldier, with "disease of the spinal cord;"

another was weak in intellect; one sister became affected about the age of 30, she had an awkward gait, and idiotical appearance, and could not hold her head still, committed suicide; another sister had uncertain gait, falling about, and progressive weakness, also committed suicide. Of the patient's seven children, one daughter is stated to have hereditary ataxy. The patient himself, a gold-worker, noticed his handwriting deteriorate from the age of 17, difficulties in walking and in doing things in the dark, from the age of 28 or thereabouts, also some awkwardness in feeding himself, and some pain in the loins, inability to pursue his trade from the age of 40; clumsiness of speech and weakness in right arm from the age of 39. When seen by Dr. Menzel first at the age of 42, head turns to right as he walks (spasm of left sterno-mastoid), facial contortions as he walks or reads: speaks with pauses and effort, as if oppressed or in pain, the longer he speaks the louder and more explosive his words become. All these spasmodic affections are remarkably improved when he lies down. Gait ataxic, with stamping and jactitation of feet, has to hold on to things. Movements of all kinds ataxic, and fine movements impossible. Patellar tendon reactions somewhat exaggerated. No pains. No sensory symptoms. Pupils normal.

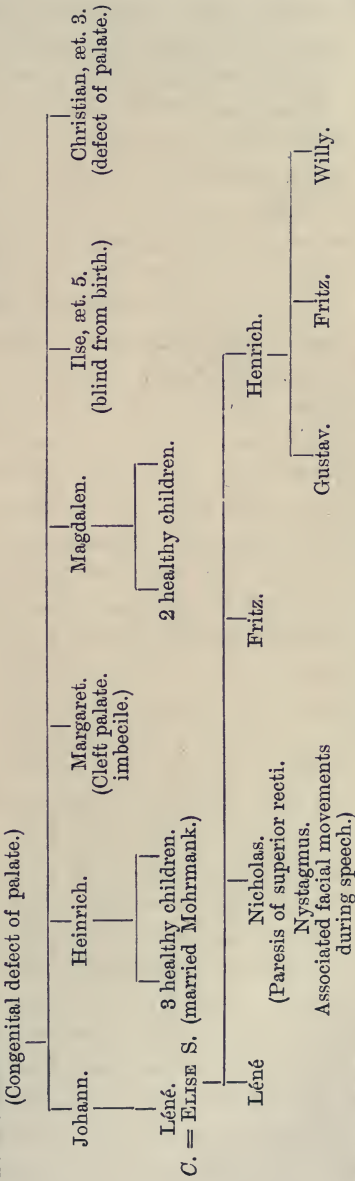
At 45 he was wasted, worn, and cachectic. There were now spasmodic movements of the jaw (=opening of the mouth), and involuntary choreiform movements of right arm. Speech still more difficult. At 46, he had difficulty in letting go of things when once he had grasped them. He died about this age. *Post-mortem* there was found (omitting some details) a noticeable atrophy of the cerebellum and pons, atrophy of the olivary bodies, and in the region of the hypo-glossal and facial nuclei. In the cerebellar cortex the nuclear layer was small, and Purkinje's cells very deficient, except in the vermis. The spinal cord was small, and exhibited degeneration of the posterior columns, most extensive in the lumbar and dorsal regions, also degeneration of the crossed pyramidal tracts, and of the cerebellar tracts.

We append Dr. Nonne's tree of the Stübe family, which shews that minor degrees of hereditary defect were widely spread among the members of it.

A. = PETER S.



B. = JOHANN S.



A, B, C. in the above table are brothers and sister.

In all the cases then, which we have examined, there was a marked heredity, and a series of symptoms which were both remarkable in themselves, and strikingly similar in the several instances. That these differ largely from those ordinarily observed in Friedreich's disease seems to us obvious. To speculate upon them seems, at present, premature. Nonne's third observation seems to show (what is indeed curious) that they may exist in the presence of simple atrophy of the nerve-centres (and particularly of the cerebellum?) without actual degenerative change. Yet to judge from the facts of Friedreich's disease, where smallness of nerve-centres predisposes to, or at least coexists with, postero-lateral spinal sclerosis, and from the last case (Menzel's) which we have given here, we shall not be surprised to find, in future *post-mortems* on cases of this kind, nervous degenerations as well as want of nervous development. Possibly the want of proper nervous development is the primary and "hereditary" factor, the spinal degeneration a special and engrafted condition, in virtue of which the case becomes relegated to one or other of the types that have been described by Friedreich and the authors quoted in this paper.

II.—BY PROFESSOR BERNHARDT (BERLIN).

It is about thirty years ago that Friedreich described a form of tabes which is developed on the basis of an hereditary, generic, or familial, basis. Erb, in his Handbook of Nervous Diseases, characterises it as follows:—It begins early, between the ages of 12 and 18, perhaps in connection with the change of puberty, or possibly with arrested development of the cord; and affects several, chiefly female, members of a family. Lancinating pains rarely exist at first; but motor troubles are very early pronounced, ataxia rapidly invading the arms, also or even attacking them simultaneously with the legs. Phonatory

inco-ordination, and atactic nystagmus. Sensory symptoms absent, or slight and very late. Cutaneous and muscular sensation long intact. No swaying on closing the eyes. No tendon reactions, bladder trouble, nor decubitus. No psychological disturbance, tremor, nor amaurosis. Paresis, contracture, atrophy, incontinence, appear very late only in this long protracted disorder (over thirty-two years).—Strümpell, in his *Manual* (Leipzig, 1884) defines the disease in similar terms, and so does Ladame (*BRAIN* xiii. p. 467), who adds to the list of pathognomonic symptoms, a peculiar club foot and frequent scoliosis. Sanger Brown's very interesting observations show that he had to do with a form of generic ataxy. But it is more than doubtful whether these cases may be classified under Friedreich's type of the disease, inasmuch as they differ from hitherto published instances in many particulars.

(1) Patients suffering from the latter disorder usually fall a prey to it early, between the ages of 12-18. In S. Brown's form this occurs sometimes early, but often much later, *e.g.*, in the forty-fifth year.

(2) Nor does the majority of his patients belong to the female sex; it is only the carrying over of the disease which happen through women.

(3) S. Brown frequently observes ptosis, which is generally absent in Friedreich's form; and universally does not note the occurrence of nystagmus.

(4) Amblyopia and amaurosis, which are never observed in Friedreich's disease, are constant and early symptoms in S. Brown's cases.

(5) In the latter the knee-jerk, which is absent in the former, is increased, and foot-clonus occurs.

(6) S. Brown does not observe club-foot nor scoliosis.

(7) In his cases the pupil-reactions are deficient; a fact not to be wondered at, since in the majority there was amblyopia and amaurosis (optic atrophy); but this symptom does not occur in Friedreich's disease.

S. Brown, at the beginning of his paper, says that if his cases are accepted as instances of this disease, it will be necessary to modify its symptomatology in various

directions; and towards the end returns to this point, on the basis of Gower's description. Erb, however, spoke¹ of two brothers, though there was no nystagmus, and though the knee-jerk was not only present, but lively. Thus whilst authors of repute regard cases as belonging to Friedreich's group, even in the absence of important symptoms, yet we have a number of observations showing us a hereditary ataxy distinct from the Friedreich type, but in several aspects similar to S. Brown's cases. P. Menzel's paper² describes the case of a man, 46 years old, with strong neuropathic inheritance on his mother's side. Out of six brothers and sisters three were affected in the same way as himself. The disease began after he was thirty years old; ataxy of the four extremities, rotation of head to the right, with associated movements in the face, speech slow and explosive, large pupils reacting sluggishly to light and accommodation, Romberg's symptom marked, knee jerks exaggerated, but no sensory troubles. Degeneration of posterior columns and roots, as well as atrophy of anterior roots, was found in the lumbar cord. In the dorsal region there was degeneration of the columns of Goll and Burdach, of the lateral pyramidal tracts, and of the lateral cerebellar fasciculi. Similar changes existed in the cervical cord. The bulb showed likewise degenerative changes; the pons was atrophic, and the cerebellum also, where the cells of Purkinje were very poorly developed. Here then we have to deal with defects, rather than with degenerations.

Nonne³ described a peculiar generic affection of the nerve centres in three brothers with one autopsy. Inco-ordination began about the thirtieth year; also phonatory disturbances, mental debility, optic atrophy, lively knee jerks; slow progress. The post-mortem evidences were very interesting. The only anomaly consisted in a general uniform smallness of all parts, not resulting from any

¹ Wanderversamml. d. S. W. deutschen Neurologen. (Baden-Baden—June 7 & 8, 1890).

² "Beitrage zur Kenntniss der hered. Ataxie," &c. *Arch. f. Psych*, vol. 22, 1891, p. 161.

³ *Arch. f. Psych*, 1891, vol. 22, p. 283. (See table, p. 277 of the present issue of BRAIN.)

inflammatory process but from a deficient pre-disposition of the nerve centres. There was no trace of the characteristic lesions of Friedreich's disease in the posterior and lateral columns; but as Nonne says, the smallness of the organs was consonant with the anatomical conditions which we are used to find in hereditary ataxy.

Schultze¹ specially called attention to the possibility of the smallness of the cord partly producing the symptoms; and Nonne (p. 314) says: The diseased condition of the optic nerve displays, as a delicate test, that there was in the case a predisposition to degenerative changes, which, however, had hitherto worked no real destruction among all other nervous structures.

I regret that pressure of work does not allow me to go more fully into these interesting questions, but from these short remarks it will appear that the apparently definite type of Friedreich's disease has been modified in various ways. In a strictly systematic sense, as the author himself allows, S. Brown's cases do not belong to Friedreich's type of disease. But if we drop this appellation, and are satisfied with such a name as hereditary, familial, or generic, ataxy, then those cases certainly fall under this interesting category, and bear certain analogies to Nonne's observations.

On anatomical grounds all these cases are characterised by a deficient development of the nerve centres, as compared to their evolution in healthy persons of similar age. This deficiency affects the cord as well as the encephalon (especially the pons, bulb and cerebellum); and in Nonne's case the peripheral nerves also. Post-mortem there may be found nothing but this abnormal smallness, but atrophies and degenerations may also occur. According to the localisation of the latter, variations will occur in single cases, or categories of cases, as to the prevalence of certain symptoms (knee-jerk, optic atrophy, oculo-muscular affections, regional muscular atrophies). The age at which the symptoms begin to manifest themselves will likewise vary from childhood or puberty to riper manhood.

¹ Virchow's "Archiv," vol. lxxix, part 1.

Whilst in one of the large groups of generic central nerve diseases, inco-ordination or ataxy plays the chief part and characterises the affection, there is another such group in which spastic elements predominate;¹ and yet a third where spinal muscular atrophies and paralyses hold the field.²

But all these morbid types, and probably also the other familial nervous affections, may perhaps, as far as autopsies go, be referred to a more or less faulty development of the centres. These defects may prevent the increased demands of advancing years upon sensation and action, from being duly responded to; or may render the structures more vulnerable to external influences than those of the normally-developed and better-resisting individual, thus giving rise to the characteristic lesions.

¹ Bernhardt; Virchow's "Archiv." vol. cxxvi., 1891.

² Bernhardt; Virchow's "Archiv." vol. cxv., 1889.

Reviews and Notices of Books.

- I.—*Il Cervelletto ; nuovi studi di fisiologia normale e pathologica.* Per LUIGI LUCIANI. Firenze, 1891.
- II.—*Sull 'origine e decorso dei peduncoli cerebellari e sui loro rapporti cogli altri centri nervosi.* Per DR. VITT. MARCHI. Firenze, 1891.

THESE two works, which are the most recent treatises on the anatomical connections and physiology of the cerebellum, form an important contribution from the modern Italian scientific school. The former gives expression to Luciani's matured views on the subject of cerebellar physiology, and includes the experiments which were recorded by him in the *Prima Memoria* in 1884. The latter records the degenerative changes in the central nervous system of the animals on which partial or complete cerebellar extirpation had been performed. It is our intention here to review in detail both these works, giving special prominence to the former, as it forms a very valuable addition to the physiology of the nervous system, and introduces a new doctrine of the functions of the cerebellum.

As the author has been very successful, both as to the number of animals (dogs and monkeys) on which he has operated, and in the completeness of the partial and total extirpation of the cerebellum, his method of operation deserves mention. Narcosis was obtained in dogs by hypodermic injection of morphia and chloral (morphia 2.5 centigram, chloral 1 gram), and in monkeys by chloroform, in addition to morphia. The muscles were separated down the middle line of the neck, and partly detached from the linea semi-circularis. An opening was then made with a small trephine in the occipital bone, and enlarged by the bone-forceps. The opening thus made extended from the external occipital protuberance to the foramen magnum, so as to expose the whole region as far as the calamus scriptorius. The dura mater was

incised over the middle lobe. *The extirpation of the middle lobe of the cerebellum* is a relatively easy operation. This lobe is separated from the lateral lobes by two antero-posterior incisions. A spoon with a cutting edge is introduced, and the brain matter removed as far as the corpora quadrigemina. When thoroughly extirpated, the floor of the fourth ventricle and the opening of the aqueduct of Sylvius are exposed. To remove *the whole cerebellum* at one sitting, proceed as above described, for the removal of the middle lobe, and then remove the lateral lobes as much as possible *en masse*. The removal of the flocculus, although not impossible, is difficult and uncertain. *One half of the cerebellum* is extirpated by dividing the vermis in the median plane, by means of a Græfe's knife. It is essential that the remaining half be not injured. It is difficult to perform hemi-extirpation of the uvula without at the same time injuring the medulla oblongata. Hæmorrhage from the brain is arrested by means of small sponges, soaked in corrosive sublimate (1 in 1,000).

The disadvantage of operating on monkeys is that the head requires to be held forward by an assistant. In favour of the use of these animals is the very ready manner in which they are narcotised, and the very slight tendency to epilepsy after the operation. In dogs this tendency is combated by hypodermic injections of chloral. The latter, also, are subject to impulsive movements after the operation, and have sometimes to be artificially fed. In monkeys, on the other hand, the irritative phenomena are less intense and more fleeting.

Having overcome the difficulty of removing the cerebellum, we must next see how best to define the phenomena which result from it. In studying the movement of an animal, several principal factors are distinguished—the energy employed in the contraction of the muscles, the tension or tonicity of the muscles, the manner of effecting the contraction, the form of the movements, their symmetry, and their co-ordination, or the manner of association, and succession of movements.

For estimating the force of muscular contraction, the author has made use of a modified dynamometer. He noticed that when a monkey was raised from the ground by means of a belt round the abdomen, it endeavoured to catch hold of any fixed object below it. He therefore attached the hook of a dynamometer to the animal's girdle-strap, raised it from the ground, and allowed it to approach a wooden cylinder maintained in a vertical position by a heavy base. To this the monkey clung with both its hands, and gradually drew out the dynamometer ring, thus enabling the

observer to read on the scale how many kilogrammes it had overcome with the arm muscles. In order to complete and control the observations on cerebellar ataxy, especially as regards the form, symmetry, and co-ordination of movement, he devised a method of tracing the footsteps of dogs on the pavement, by dipping their feet into different-coloured solutions. Throughout the work there are charts, showing the normal impress of dogs' feet when walking and when galloping, and also of those animals, at various periods after the operation, whose cerebellum had been, in whole or in part, removed.

He has studied the nature and origin of the changes consequent on mutilation of the cerebellum under five headings, and they are given here briefly to facilitate the description of the experiments to be presently noted. The theories regarding their causation are detailed later. The effects to be noted are:—

1. *Phenomena of Irritation.* These are dependent on alteration of the functions of the nervous centres provoked by the operation, and are characteristic of the first period, but it is impossible to draw a hard and fast line between these phenomena and those of later dates.
2. *Phenomena of deficiency,* depending upon deficient, unequal, or faulty cerebellar innervation. The symptoms of cerebellar deficiency are: a want of energy for voluntary acts—*asthenia*; a diminution in the muscular tonicity—*atonia*; and the presence of tremors, oscillations, titubation, uncertainty of movement, &c., or *astasia*. Here, again, one cannot separate these from the next group.
3. *The Phenomena of Compensation.* These are determined by those portions of the cerebellum which have not been injured by the operation. Two kinds of compensatory phenomena are observed: organic and functional, the former consisting of the gradual attenuation of the phenomena of deficiency, the latter, of abnormal movements, directed to correct those brought about by the deficiency. The first form is mainly observed in partial extirpation, the second in complete removal, from which we may conclude that other encephalic centres are capable of taking on the functions of the cerebellum.
4. *The phenomena of degeneration* following cerebellar extirpation are detailed when the second work is under review.
5. *The trophic phenomena* show that the cells of the cerebellum exert an influence on the general nutrition of the tissues.

§I. *Effect of division of the cerebellum into two halves.* In three animals on which this operation was performed, one dog lived twenty-two months and from it the observations are taken. There was a characteristic absence of irritative phenomena; there

was indeed tonic extension of the fore limbs, but the *post-mortem* revealed an inflammatory state of the membranes. There was, however, some difficulty in co-ordination. The phenomena of deficiency were represented by a want of energy to perform voluntary acts and a diminution in muscular tonicity and firmness. Tracings showed that compensation went on so well that in course of time it was difficult to discern anything abnormal in the gait. The author concludes that the cerebellum is not an organ which can be divided into two halves without seriously interfering with its physiological activity. It is physiologically as well as anatomically one organ.

§II. *Effects of destruction of the middle lobe.*—This lobe has generally been regarded as the most important part of the organ, especially by Nothnagel, who looked upon the symptoms of cerebellar ataxy as due to direct or indirect lesion of the worm. Vulpian's view was entirely opposed to this: "A section of the cerebellum made exactly in the median plane does not produce the same disorders of movement as lesions a little less deep, but more extensive and affecting one half of the organ more than the other." Luciani destroyed the middle lobe in four dogs and two monkeys. As an example of the effects of destruction of this lobe, I detail the observations made upon a monkey:

May 31st, 1884.—After the operation tonic contraction of the muscles of the neck was observed and tonic flexion of the upper and lower limbs. When placed in a hammock the monkey lay quiet in the supine position.

June 1st.—When offered a piece of fruit, it stretched out a tremulous arm and took hold of it, but with little energy. Placed by itself on the floor, it was unable to maintain its feet. It tended to fall from right to left.

June 2nd.—Arm still tremulous when offered food. It is apparently unable to extend the hand at the wrist, owing to tonic contraction of the flexors. When placed on a table the monkey remained with its limbs flexed, its trunk and neck continually oscillating.

June 9th-11th.—Gradual improvement in mode of walking. It proceeds with characteristic slowness, but with fair co-ordination of movements. There is still some oscillation on isolated movements. In order to test the difference of muscular energy between the arms of this animal and a healthy one, a dynamometer (as before described) was used. The monkey deprived of the middle cerebellar lobe registered 3 kilos., the healthy monkey 8 to 10 kilos

July.—The phenomena are gradually disappearing. There is still some tremor and uncertainty on isolated movements. The dynamometer registers 5.5 kilos. When offered fruit it extends its right hand, tending, however, to lay hold of some article of support with the left.

August.—All the phenomena have diminished to such an extent that there is some difficulty in distinguishing the monkey from a healthy animal.

June, 1885.—The monkey is in good health. Slight oscillatory movements are occasionally observed, which are increased when the animal is frightened. The fore limbs show a force of 9 kilos.

The phenomena observed after extirpation of the middle lobe in dogs, presented little or no variation from those observed in monkeys, with the important exceptions that in place of flexion of the fore limbs tonic extension was observed, while the general agitation immediately following the operation was considerably greater. The general features of the result of extirpation of the middle lobe are: (1) general agitation, tonic extension or flexion of the fore limbs, spasm of the neck muscles and strabismus; lasting from seven to eight days. (2) Muscular asthenia, atonia, and astasia. These are especially observed in the muscles connected with the hind limbs and are of longer duration than the irritative symptoms; they last from ten to fifteen days. (3) The phenomena of compensation ensued in all cases.

It was observed that if the middle lobe was removed perfectly symmetrically, the phenomena of irritation and deficiency were symmetrical; while if one or other side showed accentuated symptoms, it was found that the lateral lobe was injured on the corresponding side.

§III. *The effects of incomplete unilateral extirpation*, that is, extirpation of the right or left lateral lobe. The symptoms observed after this operation, which was performed entirely upon dogs, differed from those following extirpation of the middle lobe in some important points, although the general features were similar. *The irritative phenomena* consisted of curvation of the vertebral axis towards the wounded side, of tonic extension of the anterior limbs upon the same side, of rotation around the longitudinal axis from the wounded towards the healthy side and of strabismus in the same direction. The phenomena of deficiency consisted of muscular asthenia, atonia and astasia on the side of the lesion; while the phenomena of compensation took place gradually leading to restitution of the equilibrium and

ability to walk. A temporary glycosuria was observed in several of these experiments.

§IV. *Effect of removal of one half of the cerebellum.*—This operation was performed upon dogs and monkeys; the effects upon a monkey are here given in some detail:

June 26th, 1884.—Removal of the right half of the cerebellum. After recovery from the narcotism, the monkey presented curvation of the neck towards the right, the head being rotated however towards the left. When placed in a hammock there were movements of rotation around its long axis from right to left, otherwise the animal remains quiet and breathes regularly.

June 27th.—Still a tendency to rotate from right to left, but the animal endeavours to avoid this by laying hold of the network with its left arm. The head and neck are still maintained in the same position as before. The upper and lower limbs on the right side are kept in a position of flexion. The left pupil is more dilated than the right, strabismus of the left eye inward and upwards and nystagmus of both eyes.

June 28th.—Phenomena persist except that the pupils are of more equal size, and there is less rotatory tendency. When attempting to take food there is great oscillation of the right fore limb.

June 29th.—The same phenomena are present, but to a less degree. There is tonic flexion of the right fore limb, but the right leg is relaxed. The impulsion to rotation has almost ceased.

July 1.—Considerable lessening of the symptoms; when presented with a pear, it takes it in the left hand and eats it readily. It uses the left limbs, the right remaining apparently inactive. There are still oscillations of the eyes and body.

Mid-July.—Considerable improvement; it habitually embraces a small wooden column, to which it is attached by a chain, in this way maintaining a vertical position by resting on its ischial callosities. It is beginning to use the right arm more freely. The force of traction of the fore limbs, estimated by the dynamometer, is 3.5 kilos.

September.—Very great improvement; traction by the fore limbs is estimated at 8.2 kilos. When offered food, the monkey holds to the floor with the right arm, while it extends the left. Continuous and increasing capacity for walking; it raises its limbs high off the ground, but the movements are well co-ordinated. The head is still inclined towards the right, and there still remains some strabismus.

April, 1885.—It is somewhat timorous and suspicious in character; the head is still inclined to the right; when walking

the axis of the body is turned towards the right, and planting its right limbs with great abduction, a movement obliquely forwards and towards the right results. When compared with a healthy monkey, a certain indecision in its movements is observed. When offered food it invariably extends the *left hand* to take it. The pupils are equal and the nystagmus has passed off. There is still a slight strabismus shown by deviation inwards of the right eye, and upwards and outwards of the left. A slight ptosis of the right upper lid is also observed.

The symptoms of unilateral extirpation of the cerebellum therefore may be noted as follows:—1. *Phenomena of irritation* are rotation around the long axis from the wounded to the healthy side, associated with strabismus and nystagmus, curvation of the body towards the *wounded* side, tonic extension or flexion of the fore limbs and sometimes of the hind limbs. In two of the monkeys there was temporary glycosuria and polyuria. 2. The *Phenomena of deficiency* are asthenia, atonia, and astasia of the muscles on the side of the lesion. 3. *Compensatory phenomena* were observed in all cases, but a residuum of the symptoms of deficiency existed, and this is readily understood when one remembers that half the cerebellum, corresponding to one half the body, was removed, thus demonstrating that the remaining half is unable to take up entirely the function of the other.

Destruction of one lateral lobe was carried out in dogs and monkeys, which had already lost the middle lobe. The effects did not differ essentially from those which have just been recorded after unilateral extirpation.

IV. *Effects of complete and incomplete extirpation of the whole cerebellum.*—This operation was performed both in dogs and monkeys; one of the latter is given here as an example of the symptoms produced by this mutilation.

June 12, 1886.—Tonic flexion of both fore limbs, the right rather more than the left, slight convergent strabismus of both eyes. It is unable to hold itself upright. About an hour after the operation it was able to approach its back to the corner of the box which was used as a lair, and to take with its left hand a piece of fruit.

June 13.—It rests its back upright in the corner of the box and extends both hands when offered fruit. When relieved of this support the monkey falls and is unable to raise itself up again. There are oscillations of the head.

June 14.—It takes food when resting on the ischial callosities and leaning in the corner of the box. When unsup-

ported has a tendency to rotate from right to left. It can climb up the side of the cage, but cannot remain there without support.

June 16.—Placed on the floor, free from support, it takes a few steps forward, but soon falls to the right. When extending its arm to take the food offered it, it presented an uncertainty of movement not previously observed.

End of June.—The monkey now walks with greater agility, raising its fore limbs high and planting them on the floor with more abduction. There is still a tendency to fall to the right.

July.—As the monkey sits its head and trunk oscillate, but there is less tendency to fall. When eating there is continuous tremor of the arms and trunk. It walks with a hesitating oscillatory gait in a zigzag direction, tending somewhat to go towards the right. It walks more slowly than a normal monkey. During this period the general state of nutrition had somewhat deteriorated.

August.—The ataxic phenomena of the previous month have not entirely disappeared. Its general health is getting worse.

October, 1886—June, 1887.—During this period there was practically no change in the phenomena already noted. If anything, there was a slight improvement in its health.

July, 1887.—Is in a good state of health, very little change in the ataxic symptoms. When eating the monkey maintains its position by supporting itself on three limbs, using the fourth to hold its food. When resting in the ordinary way, on its ischial callosities, the hind limbs are abducted so as to enlarge its base of support. It walks inelegantly, with continual oscillations of the head, placing its limbs far apart. It proceeds somewhat obliquely with the axis of the body in the direction of walking. It descends a ladder slowly and with care.

December.—Remains the same as in the July note.

January, 1888.—The animal was killed on the thirteenth of this month. The autopsy showed that the whole cerebellum had been removed with the exception of a small portion of each flocculus, the portion on the left side being larger than that on the right. The cerebrum presented a normal appearance.

The phenomena of irritation following extirpation of the cerebellum do not differ essentially from those following removal of the middle lobe except in their intensity, persistence and greater diffusion. The phenomena of deficiency also only differ in their intensity and duration. The phenomena of functional compensation are more exaggerated.

§VI. *Effects of extirpation of the cerebellum combined with uni- or bilateral destruction of the sigmoid gyrus.*—It seemed probable

that the compensatory movements arose in the motor region of the cerebral cortex ; operations were therefore performed on the sigmoid gyrus. The operations were made entirely upon dogs, and the following is an abstract of the results obtained :—¹

The first operation consisted of removal of the right half of the cerebellum, and the symptoms were those described in section 4. The second operation consisted of incomplete and asymmetrical destruction of both sigmoid gyri. As a result there was paresis of the four extremities, associated with defective sensibility. The third operation consisted of removal of the remaining half of the cerebellum. The phenomena of irritation were shown in tonic extension of the fore limbs, curvation to the left of the vertebral axis, indicating that the extirpation was complete on the left side and incomplete on the right (and at the *post-mortem* this was shown to be the case). The phenomena of deficiency were marked and persistent ; eleven months after the last operation the dog was unable to walk without support. The conclusion to which the author comes at the end of the chapter on this subject may be expressed in his own words :—“ The compensatory movements provided for those animals which have been deprived of their cerebellum, and which have become capable of maintaining their equilibrium in the erect posture, when walking and when swimming, are dependent on the sensori-motor region of the cerebrum and they can be suppressed by simple ablation of the sigmoid gyri, which represent the most important part of that region.”

Two long chapters are now devoted to an explanation of the phenomena which we have just detailed. An analysis is first made of the irritative phenomena. The “ bilaterality ” and symmetry of the phenomena depend on bilateral and symmetrical destruction of the cerebellum. In bilateral but asymmetrical destruction or in unilateral ablation they prevail in the muscles on the side exclusively or most effectually destroyed. It is evident that these effects depend on the irritation of the efferent fibres which compose the cerebellar peduncles.

The *irritative phenomena* are : (a) pleurosthotonos, or curvation of the vertebral axis towards the wounded side, associated with tonic extension of the fore-limb on the same side, and clonic movements of the other three limbs ; (b) contortion of the vertebral axis in the cervical region, whereby the head is turned towards the unaffected side ; (c) nystagmus and strabismus, *i.e.*, deviation downwards and inwards of the eye on the side of

¹ The dog referred to here was shown at the Congress in Voghera, in 1883.

operation, and outwards and upwards on the sound side; (*d*) a tendency to rotation around the longitudinal axis following the direction of the contortion of the strabismus, *i.e.*, from the operated towards the sound side. These are the effects of acute irritation of the peduncular fibres on the wounded side. They are not continuous or inherent after the first or second day; but when the animal is approached, or taken up from its lair, or when it wishes to accomplish movements of defence, or to raise itself, or walk, these symptoms are produced. On the other hand, they become less if the animal is left undisturbed. The acute stage of the irritative symptoms gradually passes away, the tonic spasms losing their intensity and duration, and being transformed into clonic and oscillatory movements. The tendency to rotate and to fall backwards ceases, and the erect posture is possible after a short time. The capacity for walking is always re-established, but it is never possible to say that the irritative phenomena have altogether ceased. In association with the capacity for walking the animal re-acquires the attitude for floating and swimming. In monkeys, as has been shown, instead of tonic extension of the limbs, the characteristic attitude is that of tonic flexion, and further, in these animals, the symptoms are much less intense, and less prolonged, so much so that after a few days the phenomena of deficiency are most marked, every trace of irritation having disappeared.

Of the irritative phenomena one requires special consideration, *viz.*, *rotation around the longitudinal axis*. This had been observed and noticed by many experimenters, and had received various explanations at their hands. The researches of Luciani show that the rotation towards the sound side depends on the complete and successful amputation of the peduncles, the phenomenon being fleeting or partly absent when these fibres are partly untouched. It is essentially a symptom of irritation transmitted to the nervous system by the efferent fibres of the cerebellar peduncles on the wounded side.

Reference is made to two kinds of rotation: (*a*) irritative rotation, caused by traumatic or inflammatory conditions of the peduncular fibres, consists in rotation from the wounded towards the sound side, for in this instance the want of equilibrium is generated by a unilateral exaggeration of cerebellar influences transmitted by the irritated peduncle; (*b*) paralytic rotation, caused by the simple section of a peduncular strand, consists in rotation from the sound towards the wounded side, because here the want of equilibrium arises from the fact that one half of the

centres in connection with the incised peduncle suddenly lose their cerebellar influence, whilst the others retain it. But there appears also to be a form of *vertiginous impulse* which determines rotation, and this was best observed in apes after hemi-extirpation of the cerebellum. These animals learn very soon to avoid rotation, by holding on to the sides of their cage. They cling tenaciously to everything, because the fear of rotation and of injuring themselves conquers their natural timidity, owing to which, normally, they do not allow one to approach them. When placed on the floor they cling to the nearest piece of furniture, and to avoid rotation, rest on the floor not only with their body, but with their head; but after more experience they have recourse to mechanical means to counteract this tendency; that is to say, they rest on the floor with the fore-limbs spread out in abduction, maintaining themselves unmoved for an indefinite time. Anyone who watches these phenomena undoubtedly receives the impression that the monkey suffers from vertigo, and seeks in every way to avoid its ill-effects.

The phenomena of irritation having passed away, the true character of the effects of cerebellar deficiency is revealed. One important fact, which is pointed out by these experiments, is that the phenomena of deficiency are exclusively localised in the sphere of voluntary movements, and do not effect the spheres of sensation, instinct, or intelligence. Innumerable observations on animals deprived of the cerebellum have shown the integrity of the special senses, while cutaneous and the muscular sense are similarly unaffected. Further, the two fundamental instincts—self-preservation and reproduction—are unaffected by this operation. The bitches on which extirpation had been performed became pregnant, passed through the periods of labour and the puerperium with such frequency that the laboratory seemed to be transformed into a maternity hospital! The author does not believe that there is any proof of depressed psychical functions after cerebellar extirpation. In apes the natural timidity appeared increased; and in dogs he often observed during the later periods a conspicuous indolence and listlessness.

But let us now analyse the *positive effects of cerebellar deficiency*, i.e., the disordered voluntary movements which characterise animals deprived of the cerebellum. Of these there are two kinds, differing in nature and origin. They are (a) the true phenomena of functional deficiency, dependent on defective or absent cerebellar innervation; and (b) the phenomena of functional compensation represented by acts and movements,

instinctive or voluntary, by which the animal endeavours to repair the effects of the deficiency. Functional deficiency manifests itself essentially in the faulty *energy* which such animals employ in their voluntary movements, in the defective *tone* of the muscles and in the abnormal *manner* of accomplishing their contraction, while the compensatory acts are shown in the unusual *form* of the voluntary movements. Such is a brief description of the phenomena of cerebellar ataxy. Of this two typical and extreme forms can be distinguished: (1) that which follows extirpation of one half of the cerebellum, and (2) that which follows complete bilateral extirpation.

In the first division the features are of a hemiplegic nature, and are noticed on the same side as the lesion. The animal is unable to raise itself to walk, being only able to drag itself along on all fours, crawling on the buttock of the wounded side, and making the chief efforts with the limbs of the sound side. But on the contrary, during this period, the animal shows perfect capacity for floating, maintaining a good position, and of swimming with perfect co-ordination. It is constrained however, to turn towards the sound side, because it gives the water stronger strokes with the limbs of the sound side than with those on the side of the extirpation.

The dynamometric facts confirm the persistent deficiency of energy of the limbs on the side of the operation.

All the facts demonstrate that animals deprived of the middle lobe of the cerebellum, re-acquire after a certain time the capacity for walking without losing their equilibrium, this not depending on organic compensation, but upon the perfecting of certain instinctive or voluntary acts promoted by the sensori-motor sphere of the cerebrum, and determining functional compensation.

Certain of the phenomena of cerebellar deficiency which have been described have had the term *asthenia* applied to them. Conformably with extirpation of one half of the cerebellum, there is *asthenia* of the muscles on the corresponding side of the body, showing that the defective cerebellar innervation determines a *neuro-muscular hemiasthenia*. But this is not all, for associated with it is a depression of the normal tone of the muscles—a *muscular atonia*, and the following facts are illustrative of it. (a) Tremor in the muscles on the side corresponding to the extirpation, especially in the haunches during exertion. During rest or inactivity one notices greater flaccidity and less tension between the sound and wounded sides. (b) When the animal is eating, its limbs being abducted so as to enlarge its

base of support, and with its attention rivetted on its food, it has often been observed that the limbs on the defective side little by little gently flex; this produces a loss of equilibrium, and the animal falls towards the wounded side. (c) When walking there is a tendency to fall towards the defective side, which is due, not so much to inconsistent placing of the limbs of the wounded side, as to the sudden relaxation of the muscles of this side which the animal does not know how to obviate by compensatory actions. (d) Isolated elevation of the limbs of the wounded side as if the animal were ascending a small staircase.

Besides the asthenia and atonia, still another set of phenomena come under the heading of cerebellar deficiency. Such are the *tremors*, which are observed after the irritative effects have passed off. When the animal is walking quietly, they are most marked on the side of the extirpation, especially in the muscles of the limbs and of the vertebral column. They are evidence of a characteristic want of fusion and continuity of action of the muscles of the limbs and of a want of stability and firmness of the vertebral column. This also determines a certain amount of shaking, to which clinicians have applied the term, *titubation*. Under certain circumstances, for instance, when the animal is in the erect posture, or when greedily taking food, these tremors are exaggerated and assume the character of *rythmic oscillations*. The tremors, *titubation*, and *rythmic oscillations* are classified under the term *astasia*, and if limited to one side they form the condition known as *hemiastasia*.

The phenomena of deficiency following bi-lateral extirpation are diffused equally over the muscles of both sides; but there is noticed as a result of this operation a peculiar form of gait known as the drunken gait ("*démarche de l'ivresse*" of the French authors).

The phenomena of degeneration following cerebellar extirpation will be discussed later on.

The experiments show that the cerebellum is not an organ intercalated among the great paths of the cerebro-spinal system and as such transmitting voluntary impulses from the cerebrum to the spinal cord, but that it is an appendix, by the side of the cerebro-spinal paths, being essentially an auxilliary or reinforcing organ for the central nervous system, which is more developed, the higher one goes up the vertebrate scale. Complete destruction of this organ does not produce paralysis of any groups of muscles; a proof of this is found, when stimulation by electricity of the sensori-motor sphere of the cortex provokes the same reaction as if

the cerebellum were untouched. Of the greatest importance in this relation is the intimate anatomical connection which the cerebellum has with the grey matter of the pons Varolii by its middle peduncles. With this knowledge we cannot but think, that the traumatic and inflammatory irritation of the peduncular fibres is transmitted directly to the ganglionic elements of the pons.

There only now remains to briefly consider certain trophic changes. For the first few days after the operation there was noted in some of the animals polyuria, glycosuria, and acetonuria. But apart from these temporary conditions during the course of the ataxy, certain general and local trophic phenomena were observed. Marasmus was occasionally present without any evident cause for its onset. Alopecia, erythema, and eczema, were noted in several cases at various periods. Conjunctivitis, keratitis, and otitis, were observed on several occasions. It is interesting to note that these solutions of continuity readily healed under the use of disinfecting lotions. Fatty degeneration of the muscles of the extremities was also seen associated with increase in the number of the nuclei of the sarcolemma.

Several chapters are given over to detailing and criticising the various theories of the functions of the cerebellum which have been expounded since the time of Willis.

I will conclude this review by briefly referring to the last chapter of the work, which the author devotes to "the first lines of a new doctrine." The results of the researches show that the cerebellum is an organ of bilateral function, but with mainly a direct action, in this way differing from the cerebral hemispheres, which also exert a bilateral action, but mainly crossed. The experiments also indicate that cerebellar influence is not limited to the muscles which regulate the different forms of posture and locomotion, but extends to all the voluntary muscles. They further clearly show that the middle lobe is not of greater functional value than the lateral lobes, and that, as a rule, the different portions of the cerebellum have the same functions, and that the cerebellum is not a collection of centres functionally distinct or different, or that the various segments have an intimate or direct influence over special muscular groups, but that the influence which the cerebellum normally exercises over the system is a sthenic, tonic, and static, neuro-muscular action. It is also evident that the cerebellum transmits, by its efferent paths, a trophic action, direct and indirect. The direct is shown in the degenerations and scleroses which follow its ablation, the indirect by the muscular and cutaneous dystrophies which have been recorded. The functional influence, which the

cerebellum transmits normally to other nerve-centres, is slow, quiet, and continuous. But the experimental, as well as the clinical evidence, shows that in morbid states, both the trophic and functional activities are increased, so as to produce violent perturbation and disturbance of the sensorial, motor, and trophic functions. The sensorial disturbance consists essentially of vertigo, the motor of those multiform aberrations which produce motor inco-ordination, while the trophic finds expression in polyuria, glycosuria, acetonuria, and rapid decrease in weight. The trophic and functional influences probably represent different aspects of one complete physiological process, of the nature of which we are ignorant.

The functional action of the cerebellum is different from that of the other parts of the nervous system. The effects of cerebellar deficiency have been shown to be æsthenic, atonic, and astatic, while those of other centres show complete or incomplete paralysis of motion and sensation. The reason of this is that this organ forms a small, relatively independent system, defect of which does not interrupt centripetal or centrifugal conduction between the cerebrum and the peripheral nervous system. It is to be regarded as a coadjutor or reinforcing organ to the great cerebro-spinal system.

The brains and spinal cords of the animals (dogs and monkeys) on which the preceding observations had been made, were handed over to Marchi, for the purpose of following out the degenerations which resulted from the extirpation of whole or part of the cerebellum. For this purpose he used Weigert's hæmatoxylin solution, and a method which is now known as Marchi's method. Briefly stated, this consists in hardening the tissue in Müller's fluid. After a short time pieces the size of 1 c.m. are cut, and placed for eight to ten days in a mixture of Müller's fluid (2 parts) and osmic acid (1 part). The tissue is then prepared for the celloidin process. The degenerated tissue is stained black.

1. The degenerations which result from extirpation of one-half of the cerebellum (*e.g.* right hemi-extirpation) are :—

(a) In the region of the superior peduncles the osmium-bichromate method showed evidence of degeneration in the right superior cerebellar peduncle, and partly also in the left; much degeneration of the left red nucleus, and slightly of the right, while in addition there was evident alteration in the fillet, many of the fibres of the third pair of nerves, the posterior longitudinal bundle, some of the fibres of the pyramidal tracts in the crura cerebri and degeneration of the right optic tract.

(b) In the region of the middle peduncles there was found complete sclerosis of this peduncle on the side of the extirpation, the degeneration passing over the middle line. In addition there was found degeneration of the nerve fibres of the fifth pair on the same side of the fillet, of the posterior longitudinal bundle, and of a small band of fibres which lie posteriorly and externally to the superior peduncles. Sclerosis of the grey matter of the pons (nuclei pontis) was also observed.

(c) In the region of the inferior cerebellar peduncles the following degenerations were brought out by the osmium-bichromate method: The internal portion of the inferior peduncle, the external portion of the restiform body, certain of the striæ acusticæ and of the external auditory nucleus, many fibres of the ascending root of the fifth nerve, the fillet, the inter-olivary layer, and the posterior longitudinal bundle. Some of the fibres of the hypoglossal nerve and of the pyramids were also degenerated. In sections treated by Weigert's method the degeneration is not so diffuse, but there is evidence of degenerative change in the olive of the opposite side, the corpus restiforme of the same side, the direct cerebellar tract, and the fillet.

(d) In the spinal cord the author detected degenerations by the osmium-bichromate method. The Weigert method was not so successful in bringing out this degeneration. The tract in question occupies the antero-lateral region of the cord, one portion being in connection with the anterior part of the direct cerebellar tract, the other in connection with the pyramidal fibres on the same side as the extirpated cerebellar hemisphere.

As a result of ablation of the middle lobe of the cerebellum the author found the degeneration in the superior peduncles scarce and limited; but there was much alteration found in the fillet, the posterior longitudinal bundle, and the roots of the third nerves and the fibres of the optic tracts. From this one gathers that the fibres of the superior cerebellar peduncles come mainly from the nucleus dentatus and the cortex of the lateral lobes. All the transverse fibres of the middle peduncles are degenerated. The degeneration in the inferior peduncles are limited to the lateral part of the corpus restiforme. There is also degeneration of the fibres of the corpus trapezoideum and of the roots of the third, fifth, eighth, and twelfth cranial nerves. In the spinal cord there was sclerosis of the antero-lateral columns.

The author summarises his results in the following conclusions:

1. The superior cerebellar peduncles do not completely decussate, a small band of fibres passing to the optic thalamus on the

same side, the principal mass terminating in the red nucleus of the opposite side.

2. The middle cerebellar peduncles are not merely commissural strands from one hemisphere to the other. Many of the fibres enter the pyramidal bundles and end in the grey matter of the same side, while others pass to the grey matter of the opposite side.

3. The inferior cerebellar peduncles send a tract of fibres to the opposite olivary body. They are in all likelihood formed of afferent and efferent fibres.

4. The posterior longitudinal bundles and the fillet arise from a common origin in the middle lobe of the cerebellum. They course with the middle peduncles and come into relation with the nuclei of the cranial nerves, the nuclei pontis, the corpora quadrigemina, and probably also the corpus striatum. At the level of the olive the posterior longitudinal bundles fuse with the fillet and thus form a connection with the antero-lateral regions and the anterior horns of the spinal cord.

5. That the cranial nerves are closely related to the cerebellum through the medium of the fillet and the posterior longitudinal bundles.

6. The origin of the three peduncles is diffused over the cerebellum, but particularly the nucleus dentatus furnishes most of the fibres of the superior, the middle lobe of the middle peduncles.

It is impossible as yet to criticise the results which Marchi has obtained and which are briefly recorded above. The osmium-bichromate process of staining, which he has adopted, and which is now known as "Marchi's method," is, as far as my experience goes, open to numerous fallacies. The manifold connections of the cerebellum which he has described will, therefore, require ample corroboration before they can be finally accepted.

WILLIAM ALDREN TURNER.

Principles of Psychology. By W. JAMES. 2 vols., 8vo, pp. xii., 689 and 704. London: Macmillan and Co., 1890.

Text-Book of Psychology. By W. JAMES. 1 vol., 8vo, pp. 478. London: Macmillan and Co., 1892.

Prof. James' abridgment of his "Principles" is not so much a text book of psychology as a text-book of the author's psychology. So much concession he has made to criticism and general usage

that the treatment of Sensation comes first in the book. He has also taken into account the physiological ignorance of the average student of psychology, and furnished a chapter on the structure of the brain, and descriptions of the various sense-organs. The rest of the work follows, with omissions, the plan of the "Principles."

The author's working hypothesis is that "mental action may be uniformly and absolutely a function of brain-action, varying as the latter varies, and being to the brain function as effect to cause." Not that he is a disciple of the "new psychology." The question of free-will is to be ignored in psychology, being psychologically unanswerable (*Text-Book*, pp. 456, 458); but ethically the will is free (p. 461). "At present psychology is on the materialistic tack, and ought in the interests of ultimate success to be allowed full headway, even by those who are certain she will never fetch the port without putting down the helm once more" (p. 7). Psychology as a natural science is "a string of raw facts; a little gossip and wrangle about opinions; a little classification and generalisation on the mere descriptive level; a strong prejudice that we *have* states of mind, and that our brain conditions them: but not a single law in the sense in which physics shows us laws, not a single proposition from which any consequence can causally be deduced" (p. 468). Thus introduced to his science, the beginner in psychology will surely not catch the enthusiasm for it which the book of a thorough believer in the new experimental direction would give him. Prof. James himself possesses this enthusiasm in so eminent a degree that during the reading of him the paedagogic defect will not appear. But afterwards, when the factual content of the book comes to be sorted out from its setting, and the student has forgotten the phrases and images that are so telling, he will, one thinks, be either prematurely cynical or "a little mixed."

At the same time, the author is true to his plan, and the whole exposition has a neurological savour. His general attitude towards psychology differs from that usually taken up by the empirical psychologist. Instead of building up consciousness out of its units, Prof. James prefers to take the entire conscious state, as it is given, and to work at it so in the concrete. It is doubtless good that the student should be energetically reminded of the abstract nature of the "sensations" and "feelings" and "impulses" with which most of the modern books begin. But the author's method leads him at times to vagueness; to the substitution of suggestive analogy for laborious analysis; and to a fine impatience with (here and there combined with misunder-

standing of) exact experimentation. And this frame of mind, again, is not the best for the beginner.

So much by way of introduction. The chapter on "Sensation in general" bears marks of haste. On p. 14 we read: "The first time we see *light*, . . . we *are* it rather than see it." On p. 15: "The very first sensation which an infant gets *is* for him the outer universe." Which standpoint, one may ask from the beginner's point of view, is to be adopted, or are both? A similar inconsistency occurs on pp. 10, 11, with regard to the law of specific energies. Attention may be called here to the repetition on pp. 73, 413, which was probably not purposed. As to Fechner's law, Prof. James is more than sceptical. "The many pounds which form the just perceptible addition to a hundred weight feel bigger when added than the few ounces which form the just perceptible addition to a pound" (p. 21). But this is just the point: *do* they? One must hope that the student will not rest satisfied with a dictum, but will go into the matter for himself. The author's thesis that "each sensation is a complete integer" is not interfered with by Fechner's formula, as he thinks it is.

There follow chapters on sight, hearing, touch (with the temperature sense, the muscle sense, and pain), and sensation of motion. A large portion of these is adapted from Martin's "Human Body." Taste, smell and the common sensations are omitted "as almost nothing of psychological interest is known concerning them"! The whole account is rather introductory than systematic; hence such confusions as the treatment of pain under the head of sensation (elsewhere it is a primitive emotional species; p. 183), or the description of tone-pitch as a "quality of feeling." When the author comes to write his promised chapter on pleasure and pain, he will probably give a different psychological explanation of the facts of analgesia.

The sketch of the structure of the brain is clear and good; but a word might have been said as to the functions of the parts described. As it is, the chapter on brain function stands in rather loose connexion with that on structure, the cerebral cortex alone being treated at any length in the former. This is, of course, conditioned in part by the necessity of condensation; the main point for the writer being the psychological implications of brain development. And, regarded by itself, there is nothing exceptionable in the plan of the function-chapter; unless indeed it be the instance of the animal whose "life again and again pays the forfeit of his gluttony."

Under the heading "general conditions of neural activity" are brought together the subjects of nervous discharge, reaction-time, summation of stimuli, cerebral circulation and cerebral thermometry. Martius' results have not convinced Prof. James that the "muscular" reaction is not a reflex. But if this is so, how can it have a *comparative* value, for the determination of choice times, discrimination times, &c.? What we get by simple subtraction of the "sensorial" time may be doubtful (as regards certain constituents); but what we get by subtraction of the "muscular" time is a mere number, the difference of two psychological incommensurables.

There follows a chapter on habit, which is treated from a physical point of view. The discussion is exceedingly fresh and stimulating, though it is as much paedagogical and ethical as psychological, and presupposes an ethical theory. The reader will be reminded of Hering's physical treatment of memory.

It has already been said that the author advocates an analytical treatment of conscious processes, in opposition to the synthetic arrangement usual in text books. The fundamental fact for him is not the sensation or the impulse; it is "that thought goes on." Every thought is owned. No thought once gone can recur; it is the object that is got twice. ("Helmholtz calculates that the white marble painted in a picture representing an architectural view by moonlight is, when seen by daylight, from ten to twenty thousand times brighter than the real moonlit marble would be. Such a difference as this could never have been sensibly learned." The writer does not seem to see how directly this tells against his criticism of Fechner). Each personal consciousness is sensibly continuous, is a "stream of thought." Where Spencer writes that mind is composed of feelings and relations between feelings, Prof. James would say that it is made up of substantive feelings and transitive feelings (feelings to which the relations between objects are known). Every image is surrounded by a halo of relations, a "fringe." Lastly, the thinking consciousness is selective.

Psychologically regarded, the discussion of "the self" is a characteristic piece of descriptive analysis; but it trespasses also upon theory of knowledge and, *pace* the author, upon metaphysics. Noteworthy, when one considers the rigorous aloofness of the German experimental school, is the expression of his conviction that "a serious study of trance-phenomena [mediumships, &c.] is one of the greatest needs of psychology." The physiological ground of the "narrowness of consciousness" is found in the fact

that "the activity of the hemispheres tends . . . to be a consolidated and unified affair. . . . determinable only as a whole;" that of attention is threefold,—ideational and sensorial excitation of the appropriate cortical centre, afflux of blood thereto, and adaptation of the sense-organ.

Conception, the function by which we discriminate and identify, is next dealt with. The author maintains the existence of a real sensation of difference. Association is originally treated. We are to talk of association, as effect, between things thought of; as cause, between brain-processes. The elementary causal law is the law of neural habit. Psychologically expressed, Prof. James' view of "association of ideas" would be a theory of association by contiguity. *What* contiguous ideas are associated to a present one depends, of course, on many moments. Simple similars do not associate; and compound similars are partially identical. The author suggests a new nomenclature; total, partial and focalised recall; the latter representing the "association by similarity" of the older empirical psychology.

The condition of our sense of time is awareness of change. When nothing else is before consciousness, it is the organic sensations and their feelings, or the pulses of imagination and attention, that constitute the changing process which is its object. Retention and recollection are explicable in terms of the same law of neural habit which was called upon for the explanation of association. A "slumbering" condition of an organised neural path means retention; its activity means recall. The brain-tracts excited by the event and by its recall are in part different. On the other hand, the tracts for imagination and sensation are probably the same.

Sensational and reproductive brain-processes in combination give us the content of our perceptions. The process in illusion and perception is the same; what is false in illusion being not what is given, but what is inferred. The causes of illusion are ingrained association or absorbing interest. Under these conditions suggestibility obtains in all the senses. The common view that in the case of hallucination there is no objective stimulus at all is rejected, though it is admitted that centrally initiated hallucinations are theoretically possible. Physiologically considered, Binet's theory seems, I think, the more probable; but it is very difficult to get conclusive evidence.

In the chapter on "Perception of Space" the author develops his theory that voluminousness (spaciousness) is a primary attribute of sensation. He has Dr. Ward on his side;

but the view is opposed to that of most experimental psychologists. This "extensity," which is not to be confused with the psychophysical sensation-attribute of extension, is the original space-sensation (so that the title of the chapter is a misnomer); and out of it comes exact spatial knowledge, by way of discrimination, association and selection.

The chapter on Reasoning contains some suggestive remarks on animal psychology. In those following, Prof. James elaborates his thesis that "all consciousness is motor." Emotions and "instincts" are brought into close relation with one another. "The only distinction one may draw is that the reaction called emotional terminates in the subject's own body, whilst the reaction called instinctive is apt to go farther and enter into practical relations with the exciting object." As regards emotion, the at first sight paradoxical position is taken up, that the mental state results from the bodily expression. "The bodily changes follow directly the perception of the exciting fact, and our feeling of [*i.e.*, our conscious experience of] the same changes as they occur is the emotion." I paraphrase the word "feeling" as the sense seems to require; it is very loosely used in the discussion. The special nervous processes involved are sensational. The accidental factor in the genesis of the emotional reaction is strongly insisted on. Instincts are the "functional correlatives of structure." Every instinct is an impulse: is as closely connected with will, that is, as (through emotion) with feeling. Human instincts are so many that they block each other's paths; hence the characteristics of "intellectual" life, hesitation and choice. Very interesting is the author's interpretation of the two "principles of non-uniformity," the inhibition of instincts by habits and the transitoriness of instincts.

Since reflex, instinctive and emotional movements are primary, it follows that voluntary action is secondary. (There crops up at once in one's mind the opposed statement of Wundt's: "Reflexes are voluntary acts which have become mechanical.") The first condition of voluntary life is the presence of ideas of movement, memory-pictures of sensations of motion, or of sensations aroused in the special sense-organs by motion. These ideas are the "cues" of ideo-motor action, which is only another instance of the impulsive nature of consciousness. To them must be added, in certain circumstances, the "*fiat*, or element of consent or resolve," which comes in "when the neutralisation of the antagonistic and inhibitory idea is required." This *fiat*, the essential phenomenon of will, consists in the effort of attention,

and in the attitude of "consent to the reality of what is attended to." (*Principles* ii., 568.)

With the "Epilogue: Psychology and Philosophy," the introductory sentences above have already partially dealt. It contains paragraphs on the word "metaphysics," on the "relation of consciousness to the brain" and of "states of mind to their objects," and on the changing character and unverifiability of conscious states; and is sufficiently pessimistic. In one way it will be useful, as serving to remind the reader of the immense complexity of the whole subject under discussion, and so to correct the impression of simplicity apt to be left by schematic figures and explanations.

Implicitly, of course, a portion of the "Principles" has now been passed under review. Those who have read the above notice of the text-book must have felt a growing conviction that a thorough criticism of Prof. James' work must be psychological and not psychophysical or neurological, despite his natural-science standpoint and physiological theories. If this is true of the smaller book, it is much more so of the "Principles." Prof. Ladd goes, no doubt, too far, when he talks of the "exceedingly thin and dubious diagrammatic representations of brain-processes occasionally interjected into the discussion of psychological phenomena;"¹ but more than one recent work has shown how indefinite cerebral physiology still is, and I do not think that Prof. James has made the most even of the materials that are to hand. For a detailed psychological criticism this is not the place. I will only briefly refer to the chapters of the larger book, which are unrepresented in the abridgment.

In the first volume there are four such: those on the "automaton theory," the "mind-stuff theory" (partially repeated in the Epilogue to the text-book), the "methods and snares of psychology," and the "relations of minds to other things." The first-mentioned theory, according to which consciousness, at a certain stage of evolution, supervenes upon, but cannot interfere with, the sequence of autonomous neural processes, is rejected on the ground that conscious direction of the nervous system is useful. "But if it [consciousness] is useful, it must be so through its causal efficaciousness, and the automaton theory must succumb to the theory of common sense." The mind-stuff theory is also rejected, on the score of the simpleness of mental states; any "integration" or "composition" which takes place is sub-conscious and physical. (The chapter contains a good discussion

¹ *Philosophical Review*, i. 32, 33.

of the "psychology of the unconscious.") There remains the multiple-monad hypothesis, which is physiologically more than improbable; and—the soul. The author inclines to doubt the existence of the latter; though for psychological purposes he regards it as safer to rest content with the bare fact of psychophysical parallelism. Psychological method is that of introspection, experiment, and comparison; psychological errors arise from the misleading influence of language, and from the psychologist's "confusion of his standpoint with that of the mental fact" which he is regarding.

The last, rather oddly-named chapter, treats of the temporal and spatial relations of mind, and of its relations to other minds or to material things. In discussing the question: "are we ever wholly unconscious?" Prof. James adopts the somewhat crude terminology of the French writers, and speaks of the "primary" and "secondary self," of mind being "split up into separate consciousnesses," as if this were really intelligible language. The question itself he wisely leaves open.

Three chapters call for notice in the second volume: those on the "perception of reality," hypnotism, and "necessary truths and the effects of experience." Belief, like consent, is declared to be a psychosis *sui generis*. Indeed, the two are perhaps at bottom one; for "will and belief, meaning a certain relation between objects and the self are two names for one and the same *psychological* phenomenon." As experienced, belief is a "sort of feeling, more allied to the emotions than to anything else." The author digresses into theory of knowledge to discuss at length the nature of psychological "reality."

Of the three theories of the hypnotic state—animal magnetism, neurosis and suggestion—Prof. James prefers the last: "provided we grant the trance-state as its prerequisite." The chapter is not very satisfactory.

Finally, we have the chapter on psychogenesis, where Prof. James separates himself sharply from the experience-philosophy school. "The features of our organic mental structure cannot be explained at all by our conscious intercourse with the outer environment, but must rather be understood as congenital variations, 'accidental' in the first instance, but then transmitted as fixed features of the race." As regards our consciousness of classificatory, logical and mathematical relations between ideas, Spencer's views are declared "unintelligible." Darwin's explanation of the origin of instincts—by the "natural selection of accidentally produced tendencies to action"—is adopted. On

the question of the inheritance of acquired characteristics the author had arrived at an anti-Lamarckian position, independently of Professor Weismann. It is curious that the discussion moves always on the same lines, and that a theory such as that of Nägeli receives so little attention.

It is unnecessary in conclusion to express any opinion upon the general merits of the work. Prof. James' rank among the foremost of contemporary psychologists has been universally recognised. There is much in his volumes to criticise; and his attitude to the most modern and hopeful direction of psychological investigation is, unfortunately, not that of complete sympathy. But, all defects counted, there hardly exists a so stimulating and thought-compelling treatise in all the voluminous literature of the science.

The Human Mind: a Text-book of Psychology. By JAMES SULLY, M.A., LL.D. London: Longmans, Green & Co., 1892. 2 vols., 8vo, pp. xvii., 501, 393.

There can be no doubt that the recent ferment among psychologists—three important treatises, not to speak of new editions and minor works, have appeared in English within the last four years—has been conditioned to a large extent from without. The two new sciences of psychophysics and physiological psychology are the avowed *motiv* of Prof. Ladd's compilation (previously reviewed in *BRAIN*), and in the preface to the present volumes Mr. Sully makes them a determining factor in his recasting and expansion of the *Outlines*. Not that this is a sufficient description of the new text-book, which is, as the author claims, an independent work. Clearly and moderately written, and (except in one debateable regard) well proportioned, the book is a welcome addition to the literature of our empirical psychology, and will be indispensable to students of the science. If this statement seems weakened by the following criticism, it must be remembered that the psychologist and the psychophysicist cannot, in the present state of things, be expected always to think alike. There is only too much room in the sphere of mind for difference of opinion. Moreover, it is hardly possible for one man to know the literatures of three sciences equally well.

The first part of the work—"introductory"—consists of three chapters, which deal respectively with the "aim and scope" of psychology, its "data and method," and with the "physical basis of mental life." The author rightly insists upon the disparity of mental and physical processes, and upon the distinctness of psychological method. When he says, however, that "all that the scientific psychologist can mean by the phenomenal connexion of mental and bodily processes is a relation in time" (I. 47), he is, I think, ascribing to the psychologist a metaphysic which is directly opposed to his practice. Prof. Ladd, though he has gone far beyond his last, did good service to psychology in pleading for the recognition of causal interaction of "mind" and "body" as a working hypothesis in psychophysics (Elements, 648 ff). Mr. Sully cannot himself avoid dropping into terms of definite causal relation in the course of his exposition (see, *e.g.*, I. 29, 125, 135, 141, 284; II. 38, 275); and such expressions as "physiological ground," "physiological conditions" are at least ambiguous. The law of conservation of energy (II. 367-8) need not be violated by such a provisional assumption; for we may imagine the series of events to be $AB=cd=EF$, where cd is the psychical process, and yet none of the physical energy of AB is lost in EF . The logical and metaphysical difficulty of course remains untouched, but need not concern psychology.

It is difficult to say how much of the results of brain physiology should be incorporated in a psychological text-book. Mr. Sully has expressed his opinion on the subject elsewhere (*Mind*, xvi., 396). The psychologist should, on his view, "confine himself to a bare summary of the more important results of anatomical and physiological research, so far as these have a plain bearing on the understanding of physical processes, a bearing which he means afterwards to illustrate." It is no doubt unnecessary that a certain portion of physiology should be transcribed by every writer of a psychological text-book. But there is unfortunately in English no concise exposition of the physiology of the central nervous system, written by a competent hand expressly for students of psychology, such as prefaces Wundt's *Grundzüge*—now five years old. I cannot but think that such an exposition, brought up to date, would be infinitely more valuable than the main bulk of the schematic and metaphorical neurology which is to be found in these two volumes. At the same time, in the little which he has written in this introduction on the nervous system, Mr. Sully has discriminated wisely. The slender experimental basis of the theory of functional indifference of nervous

elements—probable as the theory in itself is—should perhaps have been noted. The distinction between motor and sensory cells on the ground of size can hardly hold absolutely. There are good reasons why the largest cells should be motor; but none why motor cells should not also be small. More important is the fact of the bipolarity of the cell, as established by Ramón y Cajal's researches. Lastly, the facts of localisation might have been with advantage given in a little more detail; or at least first-hand references furnished to some of the work done since 1886.

Part II. ("General View of Mind") contains four chapters, in the first of which mind is analysed into its elementary functions. The value of such analysis had been well emphasised in the introduction. Mr. Sully classifies mental functions as knowing, feeling, and willing. In all *knowing*, two stages are recognisable; the fusion of presentation with conation, and the stage of intellection proper. The latter again is three-sided, appearing as discrimination (relationing), assimilation and associative integration. A necessary condition of intellection in general is the mental property of retentiveness. The root-function in *feeling* is sensibility to pleasure and pain. The two directions of *willing* are attention and voluntary movement. All three processes are concerned in every psychosis.

This tripartite division is, as the author admits, "the outcome of subjective analysis, unaided by objective (physiological) considerations." Indeed, as regards the special function involved in intellection, Mr. Sully parts company with psychophysics. The psychophysicist, though allowing that such an analysis as the above is descriptively correct, cannot go beyond his elements; but must derive the complicated mental processes from the interaction of sensation, feeling and conation, aided by reproduction. Lewes' analysis of mental functions may stand, if for "sensible affection" we write "sensation and affection." Herbert Spencer, in regarding mind as composed of feelings and relations between feelings, saves his biological consistency by the vagueness of the word "relation," which varies between the German *Verbindung* and *Beziehung*. Mr. Sully's language tends also to be ambiguous at times; as when he substitutes the possibly passive "differentiation" for "discrimination," uses the parallels of organic segmentation and co-ordination, or talks of mental elaboration as "mechanical." In one passage the essence of the discriminative process seems to be placed in its conative side (I. 174; cf. 170). But that knowing involves a peculiar functional activity of mind which has no physiological analogue, is directly admitted for dis-

crimination and assimilation (*e.g.*, I. 171, 193, 203): though these statements again are more or less invalidated by a later one (II. 367).

The ultimate psychical elements are sensations, sense-feelings, and certain active tendencies. Mr. Sully, for the most part, sharply distinguishes between sensation and feeling, though at times he falls a victim to the looseness of the English terminology. In the note on Weber's law, Wundt's position is too closely correlated with Fechner's; it is purely psychological. A reference to Müller's *Grundlegung* for the physiological view might have been given here. The author has overlooked Aronsohn's researches on smell. Hering's theory of thermal sensations deserved a little more attention. It is surely inconsistent to make timbre a quality of *tone* sensation, while explaining that it only attaches to clangs (I. 91, 112). On pp. 112, 114, the conclusions of Prof. Stumpf's "investigations" are curiously misrepresented (*cf.* *Tonpsychologie*, II. 56, 525, 537). The connection of appreciation of direction with affection of the semicircular canals must be given up, if for no other reason, on mechanical grounds. The interaction of chromatic and achromatic qualities in colour-sensation requires more exact investigation before one is able to write so certainly as Mr. Sully does. It is not clear whether he recognises quality-differences in the *Helligkeitsreihe*. That sensory fibres are present in muscle (though not to the extent to which they occur in the tendons) may be taken as proved; though their signification for sensation is by no means clear. The author rightly rejects the hypothesis of pain-nerves (II. 14), but without referring to the best discussion of the question from the psychological side (Külpe's *Zur Theorie der sinnlichen Gefühle*). He inclines towards a belief in sensations of innervation. Here again the references are insufficient. Indeed, one may take this opportunity of saying that the space-saving method of quoting text-books, where possible, has been too exclusively followed in this part of the book. In the case of Prof. Ladd's transcriptions it becomes positively irritating. Would it not be better to refer the student directly to one or two classical researches in the various departments, from which he could find his way to the whole literature? On the other hand, Mendoza's book might have been cited, in place of Bleuler and Lehmann, for coloured hearing. The printing of "experience of movement," and "experience of impeded movement" in the text appears to imply a theory which is contradicted by the page-headings,

“sensations of movement,” “sensations of resistance.” Both views have found representatives in psychophysics. All these are, of course, minor points, and do not affect the value of the exposition as a whole.

The two following chapters deal with mental elaboration; and the first with attention. Attention in its elementary stage is described as a reflex reaction of conation upon sensation, which is accompanied and coloured by strain-sensations and sense-feelings. This is, pretty exactly, Wundt's passive apperception. That the process should be thus early emphasised and discussed is a great help to the student; and Mr. Sully's treatment is full and clear. I would only take exception to the view, that the concomitant muscular sensations can be made the measure of sensational attention; and suggest the selection of other reaction-times than those adduced on p. 155. As these stand, they are calculated to give the reader a totally false idea of the absolute duration of the simple reaction. All physiological theories of attention are as yet necessarily unsatisfactory.

In the account of differentiation (discrimination) and assimilation, Mr. Sully's language, as has been pointed out above, is not quite clear. He is, perhaps, hardly just—whether from the psychological or physiological standpoint—to those who refer our apprehension of likeness to partial identity. The psychical spring from like to like is, I think, the more “speculative hypothesis” of the two; and there is a great deal to be said in answer to Stumpf. The author recognises but one law of association of ideas: that of contiguity (I. 294 ff). There seems to be good reason for hoping that this view will soon win general acceptance among psychologists. When we have once got so far it is, more or less, indifferent whether we, for the present, describe the other constituent in the reproductive process as “assimilative revival,” (*Widerherstellung des Gleichen*), or as a case of substitution conditioned by interest, &c. A long series of experimental researches needs to be carried out, before the question as to its nature can be decided. It seems somewhat premature to regard fusion (*Verschmelzung*) as an instance of close association. The physiological basis of reproduction is, as Mr. Sully says, quite conjectural.

I have devoted so much space to the first two hundred pages of the book, because there is here given in outline the system which is elaborated later. The three divisions of the work which follow—“Intellection,” “the Feelings,” and “Conation or Volition”—present in detail the doctrine here set forth in the rough. Mr.

Sully has had it in mind throughout that he is writing a text-book. His conclusions are cautious, and will no doubt be regarded as eclectic by extremists of all psychological schools. It is, however, most important that the student should not be led "to take sides" from the outset; and the knowledge that there are two views on most psychological questions cannot be too deeply impressed upon him. The most serious blemish of the book is its hypothetical physiology. In his desire to translate psychical processes into terms of neurology, the author makes the latter vague, and even metaphorical. The reader is struck rather by the simplicity of the supposed cerebral action than (as should be the case) by its enormous complexity. Moreover, the translation has as yet, in most instances, very little scientific justification.

The section on "Intellection" begins with a chapter on perception. In quoting Helmholtz on p. 213, Mr. Sully seems to have followed Prof. James ("Principles," I. 169, II. 111) without referring to the original. The passage quoted from the *Thatsachen in d. Wahrnehmung* (1878) does not say what he makes it say.

On tactual space-consciousness he takes up a sound and moderate position, rejecting the primitive-bigness hypothesis as a psychological *ὑστερον πρότερον*. Wundt's theory of psychic synthesis is stated misleadingly as regards its intensive factor (*Grundzüge*, I. 406, II. 32. Mr. Sully repeats the mistake elsewhere; e.g., II. 212). The thirty pages on visual perception are necessarily insufficient for complete exposition. The psychogenetic child of p. 246 is open to objection. The eyelid pressure of Münsterberg and Schwarz should perhaps be supplemented by the friction of the eyeball in its socket. The time-sense can hardly any longer be brought, without reasons given, under the heading of auditory perception, especially if Münsterberg's views are accepted. The last sentence on p. 272 should be lengthened by the words "or of a common ground-tone." In the fifth, e.g., this element of relationship is important.

The following chapter on "reproductive imagination: memory" deals with the "image" (and the transition to it from the percept), "association of ideas," "representation of time," and "other forms of suggestion." The exposition is elaborate, occupying nearly a hundred pages. Here, where we are on such debateable ground, the value of the work as a text-book is especially evident. The chapter on "productive imagination" has evidently been a labour of especial love. The two final chapters of the first volume deal with processes of thought: conception, judgment and reasoning (knowledge). The preliminary account of attention renders the

discussion intelligible at this stage. Many hints are given towards a psychophysical theory (*e.g.*, of causal judgment and belief). The sharp distinction drawn throughout between logic and psychology is helpful: and the aid furnished by language to the child's attainment of an idea of self is well emphasised; it is a point too often overlooked.

The feelings are rightly regarded by the author as directly conditioned by stimulus. Whether feeling can occur independently of sensation, or sensation independently of feeling, is left undecided; though the answer would probably be negative (*ct.* II. 7, 11 with I. 77. The two thresholds are assumed to be coincident in II. 21: hence the criticism of Wundt). The physiological conditions of feeling are a state of general psychophysical relaxation, and diffuse central excitation following on stimulation of a sensory nerve. Very little is said with this: and one wonders that Meynert's theory is passed over. The existence of *gemischte Gefühle* is, I think, very problematical, as is that of emotive discharges which are wholly instinctive. The chapters on the emotions (and, in particular, the discussion of the æsthetic sentiment) are most interesting reading.

The last section of the work treats of conation or volition in three chapters, which deal with "voluntary movement," "complex action," and "concrete mental development." Primitive movements are classified as random, sensori-motor (conscious reflexes) and instinctive. It seems to be a point of honour with psychologists to have a special terminology and set of definitions in the sphere of volition. Mr. Sully tells us clearly what he means by instinct and reflex; and his remarks on impulse (*Trieb*) are especially good. His general position is that "the child finds his way to a large part of his voluntary pursuits by what we must call accidental coincidences." That is to say, given instinctive movement and sensational attention, voluntary movement will necessarily arise sooner or later under the influence of feeling. It must be noted here that "voluntary movement" has a very definite sense; volition at a certain stage is, of course, postulated with the word "attention," and the instinctive movements have themselves (as Wundt points out) the character of "simple impulse-movements." The author inclines to suppose that innervation of the antagonists plays a large part in the inhibition of voluntary action; but he does not mention Orschansky in connexion with this thesis. It is gratuitously inexact to speak of feeling at this stage of the discussion as being "concomitant *or effect*" of sensation and idea (II. 270). How absolutely conjectural the

physiological basis of volitional effort is may be seen by comparing Mr. Sully's view with that of Prof. Müller.

The book concludes with a series of fourteen short appendices mainly upon borderland questions, written from the historical point of view. The text is singularly free from printer's errors: but many words and a large proportion of the references in the notes require correction.

Handbook of Psychology. By J. M. BALDWIN. I., Senses and Intellect, 2nd Ed., 1890; II., Feeling and Will, 1891. New York: Henry Holt & Co. Pp. xv., 343; xi., 394.

That Prof. Baldwin's *Handbook* is a valuable contribution to Psychology, viewed from the educational standpoint, is shown both by the many favourable notices of it which have already appeared, and by the call for a second edition of the first volume within a year from its publication. And yet it is in this work, far more than in those previously spoken of, that the difficulty of assimilating the new manner of thinking to the old impresses itself upon the reader. One seems at times, in passing from chapter to chapter—less often within the limits of a single chapter—to spring from speculative psychology into psychophysics, without any aid from stepping-stones, and without any indication from the author that something in the way of a bridge is necessary. Prof. Baldwin will base his psychology upon “empirical investigation”: “psychometry” [why cannot we get rid of this word?], “psychophysics and neurology” shall furnish the data for inductively established hypothesis. Then why is the account of the nervous system prefixed to the second volume; to the consideration of feeling, rather than to that of sensation? As regards the physiology of feeling, in the general sense of the word (I., 108; the author is very vague about it), we know next to nothing; of the physiology of sensation we know in comparison a great deal. I will not multiply instances. The grafting process is exceedingly difficult; the more so since there are signs that psychophysics means to expand into an experimental psychology, with new concepts as well as independent methods; so that the would-be conciliator to-day is almost forced into eclecticism.

The account of the nervous system, and of its connection with consciousness, occupies the first ninety pages of the second

volume. It represents a consistent attempt at a "philosophical" treatment; physiological and anatomical details are kept in the background,¹ laws and principles of structure and action emphasised. The fundamental specific properties of nervous tissue are neurility and sentience. Of neurility there are two current views: either it is the "molecular state which constitutes a course a good nervous conductor" (electrical analogy theory); or "the nervous system is a living organism, instinct with nervous force or neural properties throughout" (dynamic theory). Whichever view we hold, the central process is the important thing; for on the latter theory the activity of every element is "central." So we come to sentience, where we must distinguish the facts of integration (law of growth of living tissue), retention and selection. Of selection again there are two views: (1) "Does the nervous system select from a multitude of similar touches? The magnet selects from a multitude of similar filings, and the explanation seems to be the same. Neither the touches nor the filings are similar after all." (2) Sentience involves consciousness, and the *differentia* of the latter is choice. Prof. Baldwin accents as strongly as Prof. James the psychological significance of the law of nervous dynamogenesis—that every stimulus has a motor force.

Nervous reaction is (1) automatic (breathing, reproduction); (2) reflex: simple (winking) and co-ordinating (walking); and (3) voluntary. Negative nervous reaction covers the phenomena of inhibition. A curious error has crept into their discussion, in that the word *erschöpft* (exhausted) has been taken by the author to mean "formed, consolidated." There are five principles of nervous action. First stands that of specialisation of function. The questions arising out of the facts of cerebral localization, are shortly treated of, partly here, partly in the first volume. Their handling is not very satisfactory. Then follow the principles of functional indifference, substitution and specific connexion. This last means two things: that the presence of definite central or [and?] peripheral connexion alone determines the specific energy of a nerve-course, and that the connexions work by way of necessitating reaction to a specific stimulus.

¹ The statement as to the relations of nerve to cell, p. 6, must be altered in the light of more recent research. Cf., *e.g.*, Kölliker, Biol. Centralblatt, 30 Jan., 1892. I cannot but think, also, that a full-page diagram of conduction-paths (Flechsig's, *e.g.*), would, with advantage, take the place of Obersteiner's schema, p. 12, and of some of the text. Lastly, is it so certain that memory-pictures form part of conscious content in the case of the brainless carp (p. 27)?

"The only specific things after all are the stimulus and the movement." The principle of summation of stimuli seems to be regarded by Prof. Baldwin as furnishing the explanation of *Einstellung*; but it is hardly adequate to this. Finally, we have the three great laws of habit (consolidation, downward growth: diffused attention), accommodation (specialisation, upward growth: interest) and inheritance (the conjoint result of the two, acting in the race).

I have analysed this chapter thus at length, because of its strictly psychophysical turn. Whether the author's revival of Lewes' terminology will find general acceptance is, I think, doubtful. It seems unnecessary to go outside the usual physiological nomenclature, and a new source of confusion is introduced by using "sensibility" as equivalent to Lewes' "sentience," and *vice versa*. In the summing up, also, an unfortunate choice of terms has been made. Habit is, anatomically considered, "the arrangement of elements more suitably for a function, in consequence of former modifications of arrangement through that function." But what is this but one kind of specialisation— which Prof. Baldwin will make the very opposite of habit?

Is the "nerve-process, irritability," conscious; or is a certain degree of development necessary for the arising of consciousness? The former view, that of Lewes, Bastian, and (in a modified form) Wundt, is preferred by the author. He finds, rather boldly, a confirmation of it in the multiple personalities of hypnotic subjects: "the higher centres being here inhibited, . . . the lower supply experience which was before outside the conscious area." But here he seems going beyond his own definition of consciousness; and this is still more clearly shown in the following paragraph, where he writes, that probably "consciousness arises from the breaking down or expenditure of the cellular structure in the highest centres." So that the acceptance of Lewes' theory halts somewhat.

Consciousness is of three kinds. Passive consciousness is "subconscious sensibility;" simple awareness, unlocalised, effortless, purely affective. Yet sensibility itself is "sentience [regarded] as a conscious phenomenon" (II. 55); while elsewhere, the subconscious area is marked off from that of passive consciousness (I. 68). In reactive consciousness we are receptive, involuntarily attentive. Voluntary consciousness is deliberative, involves effort. The three degrees of consciousness are paralleled with the three phases of nervous function; but the physiological soundness of the division seems questionable.

Further special analogies are those of nervous retention and memory; of specific connexion and association; of summation of stimuli and, *e.g.*, Weber's law; of inhibition and the Herbartian idea-conflicts, or the mental processes consequent on suggestion. There follows a criticism of Dr. Maudsley's theory of the unity of consciousness, which is substantially a reproduction of an article published in *Mind*, in 1889, and some short remarks on mental heredity.

Another instance in this volume of the author's method is his discussion of theories of the sense-feelings, pleasure and pain. There are three groups of such theories; the static or absolute, the dynamic or relative, and the genetic. Physiologically, the former finds its expression in Mr. Grant Allen's definition of pleasure as "the subjective concomitant of the normal amount of function in any sentient tissue." Intellectually, it relates feeling to knowledge; and it is here that Prof. Baldwin parts company with it. In ruling out the Herbartian view, that "makes feeling dependent on the intellectual function considered as a play of representative elements or forces," on the ground that a sharp distinction of sensation from feeling is inadmissible, he is at variance with current psychological thought, which insists on just this distinction as primary. The second view, that of Ferrier and Dumont, says that all expenditure is painful, and all integration pleasurable. This the author meets by reference to the pleasure of novelty, which means pleasure in increasing expenditure. The genetic view is practically that of Aristotle. According to it, pleasure is the "conscious effect of that which makes for the continuance of the bodily life or its advancement." Is there not here again a mixture of psychological description, based on introspection and reflection, with psychophysical explanation? And does not Prof. Baldwin fall into the same error, as regards feeling, which "even the biologist Schneider" avoided, as regards consciousness in general?

In the remainder of the volume, which deals with the ideal feelings, and with the phenomena of will, there is much acute thinking, and a good deal that is original. The "Senses and Intellect" follows a more beaten track. After the introduction come two chapters on "general characteristics of mind," the second of which deals with Attention. It is a great gain to the student here, as in Mr. Sully's work, to have the latter process treated, at least in outline, thus early. The second part of the volume deals with the apperceptive function (presentation, representation, combination, and elaboration) and the rational function.

In the treatment of sensation, extension (*Ausdehnung*) is not included in the list of attributes (I. 85; but. cf. 109). The author decides in favour of the physiological interpretation of Weber's law, but, strangely enough, does not mention G. E. Müller in connexion with it. The account of chronometrical results is hardly adequate. One has no wish to carp upon this matter; but it is surely misleading to give .125" as simple reaction-time, without naming the sense for which it holds, or stating that it represents the "muscular" form. The number itself seems to point to hearing-experiments. The account of the "theory of unity of composition of mind" would be better placed after the discussion of the organic theory of mental unity in the second volume.

Prof. Baldwin has taken a wide view of his science. He includes not only the two psychologies proper, empirical and rationalistic, but deals, in connexion with them, with logic, theory of knowledge and metaphysics. With those who hold that there is but one psychology, the empirical and experimental, he would agree to differ; they do not constitute the tribunal whose decision upon his work he would accept. A detailed criticism of his theories of sensation, &c., is therefore unnecessary. More serious are the objections which affect arrangement and terminology, and which have been already urged by other reviewers. Both faults can be corrected in future editions, and the Handbook will thereby be rendered even more valuable to the general student of psychology than it now is. An especially useful feature of the work is the list of "further problems for study," appended to each chapter.

An Introduction to Human Physiology. By A. D. WALLER, M.D. London: Longmans, Green & Co., 1891. Pp. x., 612.

About half of Dr. Waller's book—eight chapters—is taken up with a consideration of the phenomena of excitation in the human body; and it is with this portion of his work that we are here concerned.

The author begins his exposition with a chapter on the general plan of the nervous system, in which he shortly illustrates the progressive elaboration of the neuro-muscular apparatus from the *amœba* to man. The nerve-centres in man are classified as.

cortical and medullary or spinal. With the former are correlated voluntary actions, with the latter automatic and reflex actions. This correlation is, indeed, admittedly imperfect and, in special instances, arbitrary; for the physiologist is in no better case than the psychologist as regards a strict definition of the terms "reflex" and "automatic." In describing an automatic action as "a series of immediate motor reactions," Dr. Waller includes under it on the one hand such acts as breathing (primary automatic action) and on the other such acquired motor co-ordinations as are called into play in writing (secondary automatic actions). Such acts as walking occupy an intermediate place between the two main groups. In classing all these functions together the author varies from ordinary psychological usage, but is enabled to avoid the doubtful word "instinctive."

At the conclusion of the chapter is placed a paragraph upon the limitations of scientific enquiry. "The question *why*? is not answered by positive science, but only the question *how*? and sometimes the question *how much*?" It is quite true that no science can explain its first principles; and quite true that in the particular case before us the transition from "physical" to "psychical"—*e.g.*, from æther-wave to sensation—is wholly inexplicable. But one must not go too far. A proximate *why* is given by science in every case where she gives a more general *how*. The proximate *why* of human embryology is furnished biogenetically. In this sense the physiologist *can* "say why a muscle contracts," if he brings muscular contraction under the laws of pyro-electricity, *e.g.*:—by the way, there is no mention of Prof. Müller's theory in the chapter on muscle:—though for the physicist it is still only a matter of *how*.

The two following chapters deal with the peripheral nervous system, under the aspects of muscle and nerve respectively. "Muscles may indeed properly be regarded as the end-organs of motor nerves." This grouping is convenient; and though, perhaps, at first sight likely to mislead the beginner in physiology, will not cause any permanent misunderstanding, after the contents of the chapters have been digested.

After a useful description of electro-physiological instruments, and a short account of the histology and chemistry of muscle, the author proceeds to deal with its physical and physiological properties. For the study of the so-called muscular sense it is of the utmost importance to have in this regard an adequate and definite terminology. To judge from the footnote on p. 324 this seems to be as little the case in English as it unfortunately is in German. According to Dr. Waller, muscle is, physically re-

garded, extensible (*i.e.*, can be elongated by weighting, &c.) elastic (*i.e.*, shortens after removal of the weight), and viscous (*i.e.*, does not shorten to its unweighted length immediately, on withdrawal of the weight: the direct elastic shortening is followed by a gradual indefinite shortening). Physiologically considered, muscle is excitable; and this excitability manifests itself as contraction. It would, I think, be better here to use the word "excitation" and not "contraction" as the general term. A muscle may be excited in circumstances where contraction is impossible. But the main thing is to call the student's attention to the mutual independence of the conditions of muscular tension and excitation: and this the author does. In the rest of the chapter the main features of muscle-physiology are treated of. There is a good paragraph on the mechanics of muscular arrangement.

Dr. Waller is justly sceptical as to the existence of "pathic" nerves. He would, perhaps, have spoken still more decidedly, had he looked at the question from the psychophysical side, and realised what the hypothesis must mean, when every separate quality of the tone or colour scale can, and does, if carried beyond a certain intensity, pass over into pain. His remarks on "trophic" nerves are a model of careful reasoning. On p. 351, in connexion with the law of specific energies, a reference forward to p. 375 (where the functional indifference of the conductor is emphasised) would perhaps be in place. To the experimental proofs adduced for the latter might be added Babuchin's experiment on the electric nerve of *Malapterurus*: and du Bois-Reymond's name should have been mentioned. The exposition of the phenomena of electrical nervous stimulation is admirably clear; indeed, the author's account is by far the best of its compass with which I am acquainted in English or German.

A brief chapter on animal electricity gives a useful *résumé* of facts and theories from Galvani and Volta to du Bois-Reymond and Hermann.

There follows a more detailed review of the special senses. Curiously enough, Dr. Waller chooses to deal with colour entirely under the heading of the "physical data" of vision, though aware of his inaccuracy (p. 401). What he writes, however, is so much to the point that one can almost overlook this. His remarks on saturation and intensity of colour are not quite clear. He appears to maintain (what is not improbable) that colour-saturation depends on the amount of white light with which the colour is mixed: colour-intensity on the intensity of this white light. If this is not the sense of the passage, there is a con-

fusion between the physical and the psychophysical (or physiological!) aspects: if it is, it should be definitely pointed out that colour-intensity may be regarded as a function of saturation, and that the colour-scale presents sensational differences which are qualitative only. This is, indeed, plainly enough hinted by the diagram on p. 404; where a red is shown to be "dilute" or "dark" according as it is mixed with different quantities and qualities of the brightness scale.

Wundt's theory of colour-vision should have been mentioned on p. 407, if only to illustrate the all too suggestive fact that the progress of such theories has set steadily from the concrete to the abstract. A word might have been said, also, on the errors of perimetric experimentation. Moreover, as the sentence on p. 408 stands, it contains a direct contradiction of Hering's colour-theory (p. 407), which may lead the student astray. The paragraph on corresponding retinal points and on the horopter seem a little uncritical. The author is throughout specially happy in his diagrams. Fig. 223, p. 429, which illustrates the action of the muscles of the eye-ball, deserves in particular to be copied into all future physiological and psychological text-books. E. Fick's replies (1889, 1890) to the investigations of van Genderen-Stort and Engelmann have apparently not been noticed (p. 425). For binocular contrast the reader is referred to Hering. This is an error: Fechner's elaborate discussion was published in 1860, and the first experiment quoted is taken from him.

Much that is psychophysical is included, again, under the rubric of "physical data" in the case of the ear. I am very glad to see that Dr. Waller roundly rejects the hypothesis that the semi-circular canals are concerned in the appreciation of the direction of sound. He proposes a modified form of Rutherford's theory of hearing (independently arrived at) in place of that of Helmholtz. What is wanted here is facts; and especially facts relating to the mode of vibration of the auditory hairs. Nothing is said of a theory of taste or smell, or of the temperature-sense: and the treatment of the "muscular sense" is unsatisfactory, no reference at all being made to Goldscheider's work on articular sensibility. The incorrect statement made in this connexion about Wundt has probably been taken from an early edition of the *Physiologische Psychologie*.

The two final chapters deal with the spinal cord and bulb, and with the brain. The evidence relating to sensory and motor conduction-paths in the cord is carefully sifted. Good remarks are made upon the "centres" of the cord and the medulla; as regards which the beginner (I can speak from experience)

is especially likely to go wrong. The possibility of the presence of retino-motor fibres in the N. opticus might have been alluded to. The discussion of the facts and hypotheses of cortical localisation is excellent. The student is impressed with the enormous complication of the subject, and with the need of extreme caution in drawing inferences, at the same time that data are furnished him for an appreciation of the value of conflicting methods and statements. The author recognises the unsuitability of Munk's nomenclature, and is praiseworthily careful in regard to Jackson's schema. In the paragraph on reaction-time, the influence of the direction of attention is noted; but it should then be stated that the cases given in fig. 279 illustrate the "muscular" form. The times adduced for touch and hearing are surely too long. In the statement of the Weber-Fechner law it should be insisted that the "least observable differences," or sensation units are only regarded by Fechner as equal in their subjective contexts, and in the absence of all opportunity for objective measurement or estimation. Dr. Waller plainly means this, but says it rather curtly; and there has been much misunderstanding of the point.

The explanation of the optical illusions on p. 541 is too short; it credits the reader with certain psychophysical knowledge. The question as to the nature of colour-contrast (for the answering of which Kirschmann's work should have been taken into account) is left just where Fechner left it thirty years ago. I do not mean this as a reproach; it may be that the answer given is correct; in any case it is, in this instance, based partially upon the author's own experiments. But one should certainly take some account of binocular contrast, in seeking a plausible hypothesis; and if one inclines, in whole or part, to a psychological interpretation of the phenomena, one should avoid the word "judgment." The chapter concludes with some very sensible paragraphs upon hypnosis.

So far as it has been here examined, this book is to be cordially recommended. The style is lucid and simple; and Dr. Waller handles "frontier questions" with a modesty and soundness which, if common, would soon put an end to the mutual distrust that, unfortunately, so often exists between the physiologist and the psychophysicist. It remains to be said that the rest of the work consists of seven chapters on the phenomena of nutrition; three appendices (on the origin and nutrition of the embryo, on constitutional formulæ of proximate principles, and on units of measurement); a confessedly incomplete, but useful bibliography; and a good index.

E. B. TITCHENER.

Contribution a l'étude anatomique et clinique de l'Acromégalie et en particulier d'une forme amyotrophique de cette maladie. Par le Dr. G. DUCHESNEAU, ancien interne des Hopitaux et de la Maternité de Lyon. Chap. vii., pp. 206. Paris : Baillière et Fils, 1892.

Dr. Duchesneau admirably justifies the title of his monograph: his contribution is truly a study, and a valuable addition to the now extensive literature of the subject. From the number of cases reported, and the careful analysis of symptoms, we can strongly recommend its perusal to all who wish to investigate the disease.

The first chapter summarizes the previous literature of Acromegaly, and briefly traces the gradual separation of this disease by Marie from Paget's osteitis deformans, Virchow's leontiasis ossea, myxoedema, and other diseases all having a strong family likeness. It is also mentioned that in 1890 Marie, from a careful examination of M. Gourand's case, recognized "hypertrophic pulmonary osteo-arthritis" as a disease having a definite causation, and distinct from Acromegaly with which it had before been confounded. Dr. Duchesneau points out that although since Marie's original essay and M. de Souza Leite's thesis many clinical cases, reviews and anatomo-pathological papers have been published, most attention has been paid to the theoretical side of the question, and expresses his desire to treat rather of nosography and pathogeny.

The second chapter contains 31 collected cases, nearly all recorded since the publication of M. de Souza Leite's thesis; they are carefully and conveniently arranged into three groups, 1st (14 in number), comprising observations sufficiently complete for anatomical or clinical study; 2nd (13 in number), cases only shortly detailed; 3rd (4 in number), cases described as acromegaly, but not really examples of that disease. Of these latter the author considers that three were probably cases of hypertrophic pulmonary osteo-arthritis, the fourth is rejected from the early age of the subject and the absence of any enlargement of the inferior maxilla.

The third chapter shortly refers to ætiology. With M. de Souza Leite the author admits how little is at present known of the causes of acromegaly but gives the following results of an analysis of the cases collected by himself, together with those of M. de Souza Leite.

That acromegaly is slightly more frequent among females than males, numbers being 30 and 23 respectively. That it is not congenital.

That nervous affections, including painful moral impressions and psychoses, may act as causes, "soit predisposantes, soit même déterminantes." Dr. Duchesneau remarks it is curious to note that in no case among his own collection, and only in three of M. de Souza Leite's, is there any history of syphilis or alcohol.

The fourth chapter treats of symptomatology. A rapid sketch is first given showing what is the type *le bloc* of acromegaly. Secondly, the groups of symptoms are given in detail and put in relief. Thirdly, Dr. Duchesneau's own case is given, the symptoms analysed and compared with those found in other cases. It is pointed out that there is at least a form of acromegaly which, from the striking alterations in muscles, may be qualified as "myopathique."

Dr. de Souza Leite's method is followed by Dr. Duchesneau in rapidly sketching the symptoms which are therefore classified, thus:—

1. CONSTANT OBJECTIVE SYMPTOMS.—*Hypertrophy* of the *extremities* of upper and lower limbs, especially of the hands and fingers, feet and toes, all the tissues being implicated and giving an appearance which has been compared to paws or battle-dores.

The dimensions of the wrist and forearm are usually unaltered, those of the ankle and leg are less frequently normal. But as Marie has remarked the greatest change is seen in "*the bones of the extremities and the extremities of the bones.*" In the *head*, the cranium is little altered, thus forming a striking contrast to the face which is oval and elongated vertically. The nose, lips, especially the inferior and the lower jaw attract attention at once, all being immensely enlarged, the latter giving a massive prognathism to the face. The tongue is usually much enlarged.

In the *trunk* the spine and thorax are affected, the former presenting a kyphosis in the cervico-dorsal region from hypertrophy and deformity of the vertebræ; the latter projecting from deformity of clavicle sternum, ribs, &c., forms with the spine a "double hump" (Marie).

2. INCONSTANT OBJECTIVE SYMPTOMS.—An enlargement and thickening of the neck; an enlargement of the larynx; atrophy of the mammary glands; a pendulous condition of the abdomen; hypertrophy of the genitals; varicose veins; phosphaturia, albuminuria, &c., &c.

3. CONSTANT SUBJECTIVE SYMPTOMS.—Marked cephalalgia; amenorrhea, without loss of sexual power. Alterations in special senses, especially sight. Exaggerated appetite and great thirst.

4. INCONSTANT SUBJECTIVE SYMPTOMS.—Alteration of hearing, smell, taste; palpitations of the heart; severe abdominal pains simulating colic.

5. GENERAL SYMPTOMS.—Lassitude, disinclination to exertion. Mental depression. After giving at great length the symptoms of the head, the trunk, the limbs and the various systems, Dr. Duchesneau gives in detail his own case, and notes the points of agreement or disagreement with his fourteen collected cases as well as with the classical type of acromegaly. Dr. Duchesneau's case was under observation for fifteen years, and was an undoubted example of the disease, but is also exceedingly interesting and noteworthy, clinically and pathologically, for reasons of neurological and general interest. It is only possible here to enumerate the principal points: the onset, with acute pains in various regions, head, spine, limbs, &c., with loss of power; the presence of hypertrophy, obtaining in the usual situations; the continuance and gradual increase of the pains and amyotrophy, gradual loss of acute vision, passing attacks of œdema at the crisis of the pains; enlargement of the thyroid gland; the presence of a floating kidney; long-continued *phosphaturia*; final termination by nephritis. After carefully analysing all the symptoms Dr. Duchesneau draws special attention to the very advanced muscular atrophy, and quotes the cases of Godlee, Gabian and Claus to support his contention that there is an amyotrophic form of acromegaly. To the amyotrophy is attributed, partially at any rate, in addition to the loss of power in the limbs, the marked kyphosis, the chest deformity, and the floating kidney.

This long chapter, too long we think, is ended by a consideration of other clinical forms. Dr. Duchesneau points out the disproportion in various cases between the ocular troubles and the duration of the case. He does not agree with the hitherto generally accepted view that the hypertrophy of the hypophysis cerebri will always account for these troubles, and therefore suggests as another clinical form of acromegaly a type characterised by alterations of visual apparatus.

Finally, a juvenile form, with general increased growth, is mentioned, but not enlarged upon.

The fifth chapter treats of pathological anatomy, being an account of the lesions found in Dr. Duchesneau's case and their comparison with those which have obtained in other cases. The constant pathological lesions are termed the "*Triade hypermégalyque*," viz.: lesions of the skeleton, integuments and hypophysis cerebri. There are also less constant lesions found in the

various systems. Dr. Duchesneau's case agreed in most points with the classical type, but there are several very important variations. Moreover, a microscopical examination of the bony tissues has elicited some striking facts bearing on the pathology.

The *cranium* presented more changes than usual. These Dr. Duchesneau attributes to the advanced stage of the disease. The sutures were almost obliterated; the parietal and occipital bones of uneven thickness; the superior maxilla much hypertrophied. The *spine* presented no lumbar lordosis. On opening the spinal canal there were no inflammatory adhesions of the dura mater, but the canal throughout was lined with a remarkably thick layer of "*cellulo-adipose*" tissue, which appeared to belong to the periosteum. It was in some places a centimetre in thickness; passing through the articular foramina of the vertebræ it compressed the nerves, which, even to the naked eye, were evidently altered. And it was just those nerves supplying the atrophied muscles, which were most altered. Microscopic examination of their roots showed disappearance of axis cylinders and segmentation of the myelin. These lesions thus account for the neuralgic phenomena and the muscular atrophy.

Dr. Duchesneau ably demonstrates by a consideration of sections taken from various bones that the increase in their size is always due to an increase in the spongy tissue, which has taken place at the expense of the compact tissue, so that there is a remarkable increase in the number and extent of the medullary spaces. This change takes place very gradually, and is not inflammatory, as after a prolonged and careful examination no giant cells, myeloplaxes, or Bizzozero's cells, with budding nuclei, were ever found. Therefore this condition differs from osteitis, and also from that of young growing bone; it differs also from rickets where the increase in size of the bone takes place in the cartilage, periosteum and outer compact tissue at the expense of the medullary portion. Again the changes in acromegaly are slow, regular and continuous, while in Paget's disease a tumultuous heaping up of the spongy tissue takes place, and isolated fragments of bony lamellæ are found, being the remains of broken-down Haversian systems. Lastly, Dr. Duchesneau remarks that all the bones affected normally contain red marrow, marrow, that is, remaining embryonic.

The changes described in the integuments are analogous to those mentioned above as found in the bony-tissues, viz., an increase of fibro-adipose tissue occurring slowly and without inflammation so that there is a real hyperplasia. The orifices of the sebaceous hair follicles were enlarged and their secretion had been increased. The nails were much striated. The increase in size of the tongue was due to increase of "*cellulo-adipose*" tissue; the muscles were not affected, there were no distended lymphatic spaces as in true macroglossia, the mucous membrane was healthy.

The hypophysis cerebri, owing to its fragile condition, could not be examined.

A systematic examination of the brain and cord has yet to be made.

There was a persistent thymus, normal in structure though hypertrophied. The enlargement of the thyroid was due to a cystic degeneration in some parts of which ossification had taken place; it closely resembled, under the microscope, an alveolar carcinoma. And attached to the first two ribs on the left side were two tumours of the same structure, each about the size of a fowl's egg.

Floating kidney was found as diagnosed and also nephritis accounting for death.

The sixth chapter contains some general observations on acromegaly. The principal theories which have been propounded are shortly referred to. Freund regards acromegaly as an *anomaly of development*. Klebs on the strength of a persistent thymus gland has constructed the theory of *l'angiomatose thymique*. Holschewnikoff and Recklinghausen elaborated a *nervous theory*, thinking that a lesion of the central and peripheral nervous system will account for the disease. Marie has connected acromegaly with *enlargement of the pituitary body*. Dr. Duchesneau does not accept any of these theories, there being a want of evidence in support of them. Returning to his own case he proceeds to connect the systematic hyperplasia of spongy tissue, in bones having red marrow, with the phosphaturia observed during life; and shows by a chart that at special crises, when fresh changes may be supposed to have been going on in the bones, there was an increase in the amount of phosphoric acid eliminated, thus indicating the onward march of the disease. Next Dr. Duchesneau suggests a relation between the bony lesions, the fibrous tissues inclosing them and the integuments surrounding both. He supposes that the connective tissue bundles of the periosteum when no longer taken up to form fibrous tissue as the fibres of Sharpey simply develop into ordinary fibrous tissue, that the blood vessels of the periosteum, no longer used to supply compact growing bone degenerate into fatty columns. Thirdly several factors are suggested as taking part in the hyperplasia of the integuments. A stretching of the tissues by the enlargement of the bones. A hyperplasia from chronic venous and lymphatic congestion, &c. In chapter seven various methods of past treatment are reviewed and the opinion expressed that in the present state of our knowledge treatment can only be symptomatic.

From this brief resumé it will be seen at once that the value of Dr. Duchesneau's exact and careful observations needs no accentuation. The changes noted in bony tissue doubtless occur in all cases of acromegaly, and the amyotrophic form is well accounted for by the nerve changes found in Dr. Duchesneau's own case. The cellulo-adipose tissue derived from the periosteum in its turn seems to have caused the nerve lesions. Thus the chain of events is complete so far, and we hope, with the author,

that other parts of the pathogeny of acromegaly will in like manner soon be cleared up.

We await with interest the report of the systematic examination of the brain and spinal cord, which has yet to be made. The author makes no mention of the sympathetic nervous system, the hypertrophy of which was regarded by Marie as being absolutely constant in acromegaly, but perhaps he will report on this also with the other systems. It is important that this should not be overlooked in future autopsies.

The possible part played by the amyotrophy as a factor in the production of the bony deformities, kyphosis, talipes, &c., and also by relaxing abdominal pressure of the dislocation of the kidney is an interesting theory, but we think Dr. Duchesneau has not quite established it. For kyphosis, with deformity of the vertebræ, is a constant symptom, while amyotrophy, at any rate a considerable amount of it is inconstant. Again, floating kidney is fairly common in women, especially in multipara, although laxity of the abdominal walls is doubtless a factor in them, there is no large amount of muscular atrophy.

Dr. Duchesneau advances good grounds for recognizing the existence of a form of acromegaly with prominent ocular troubles, and expresses his opinion that the mechanical action of the enlarged hypophysis is not the sole factor in its production, although he does not suggest any others. Possibly the narrowing of the optic foramina, caused like that of the vertebral foramina by bone changes and compression by the periosteal cellulo-adipose tissue, may be one of the factors in such cases.

With regard to the general aetiology we would advise caution in accepting, without weighty evidence, depressing nervous influences, the psychoses, &c., as causes, since histories of such troubles are not only very common in various diseases but so easily obtained that not much reliance can generally be placed on them. The presence of mental depression in acromegaly is often explained we think by the pressure of the tumour at the base of the brain.

Lastly, treatment so far can be only symptomatic, but may not the phosphaturia which probably varies directly with the rate of degeneration in the bones at some future time be a guide to treatment. If not itself to be treated it will possibly indicate the rapidity of the disease and thus aid in its regulation.

Meanwhile Dr. Duchesneau is to be congratulated on his very excellent monograph and the progress made in the pathology of acromegale from his careful investigations. As Virchow has said "we are here engaged on a most obscure subject which probably only in the course of several years will be perfectly elucidated through new observations." And it is by such accurate and extended observations as Duchesneau has made that we may expect to finally clear up the subject.

ERNEST WILLS, M.D.

BRAIN.

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Original Articles.

ON THE FUNCTIONAL ATTRIBUTES OF THE CEREBRAL CORTEX

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§ 1—INTRODUCTION.

THE following pages¹ embody an attempt in some degree to estimate the functional significance of the experimental data acquired during the last two decades on the cerebral cortex.

The immediate occasion of the attempt was a study of the "sense of effort" which has occupied my thoughts at intervals during the last few years, and has inevitably connected itself with reflections upon the psychophysiological

¹ This paper is in the same grooves of thought as a paper published in *BRAIN* last year on the Sense of Effort.

It contains in fact most of the considerations that form an area of indistinct consciousness surrounding the area of more distinct consciousness relating to the Sense of Muscular Effort.

The two affirmative propositions—"This is a fact," "I think this is a fact"—bear widely different significations in common language, but are psychologically of identical meaning; they give expression to an identical psychological conclusion, and in scientific analysis as distinguished from special pleading the first assertion, however clearly and dogmatically it be propounded, can never carry more weight than the second; conviction in the mind of an onlooker or listener should depend, not upon the sense of certainty felt to be possessed by the utterer of a conclusion, but upon the verifiable premises of that conclusion exposed to critical examination.

I have attempted to make some distinction between the more psychological and the more physiological aspects of the question; a satisfactory distinction has been impossible, physiological descriptions necessarily

interpretations that have been given of cortical activity, more especially as presented in the writings of Hitzig, Ferrier, Munk and Bastian. It has no less inevitably connected itself with purely psychological considerations, as presented more especially in the writings of Bain, Wundt and James.

These are indeed by no means a complete list of the considerations and data underlying the particular topic of study designated by the title "sense of effort"; the significance of the phenomena of attention, and more especially their relation to "motor" and "sensory" events continually presented itself; the phenomenal content, common to the psychological terms—"will," "memory," "effort," "attention," had to be reckoned with; the everlasting and never solved problems coming to our minds under the various verbal labels "free will," "determinism," "predestination," "fatalism," "moral responsibility," "automatism," &c., occasionally obtruded themselves.

I allude to these ultra-scientific matters, only to say that, in my opinion, the superficial logical connection between the study of neural phenomena, and an "*explanation*" of the final causes of neural processes, by animistic references to "force," "matter," "will," "faculties," "mind," in short by any kind of disguised anthropomorphic materialism, is in relation to positive knowledge neither scientific nor ultra-scientific but anti-scientific. Beyond every "*explanation*" we are faced by the insoluble mystery, confessed but not

include psychological inference, psychological descriptions are in part based upon physiological phenomena, seen, reported or imagined. The attempt has, however, led me to separately present in § 4 a least positive group of considerations which, although I have not been able to omit them altogether, can be omitted in the reading without prejudice to the main argument. In common with many thinkers, I regard logical forms of verbalisation as an outward and visible sign of the inward processes of ratiocination which are functional attributes of the cerebral cortex; this belief has to a considerable extent governed my mode of thought, and I could not suppress § 4 without committing a reticence of exposition. I am as sensible as the most exacting critic can be of the unsubstantial and uncertain character of any inferences from external formulations of thought, to the hypothetical neural processes of which sensation, perception and ratiocination are the subjective concomitants.

explained in the words:—"It is the spirit that quickeneth," recognised but not comprehended in Aristotle's conception of the immaterial Psyche—nutritive, sentient and intelligent.

Therefore, in spite of its superficial logical consequence, I shall premise, as a fundamental postulate, that voluntary movement, objectively studied, is reflex movement. It is no addition to description, and, although at times convenient or even necessary, in many instances it serves to arrest description, to refer voluntary movement to Will. What is Will? What is Force? What is Matter? We do not know. Our business as students of nature is with the relations between phenomena—subjective phenomena in our own consciousness mentally projected outside our own consciousness, and as a general working hypothesis, believed to be the effects of corresponding objective phenomena around us. What is consciousness? We do not know.¹

But assuming the real existence of ourselves and of our surroundings, we may, having recognised that we do not actually *comprehend* the verbo-mental images—Force, Matter, Motion, investigate and describe the phenomenal relations which, for convenience, we attribute to these elementary notions. Description comes to its dead end when we have traced back the phenomenal sequence to that point where we find it necessary to evoke an imaginary substantive force. A neural process for instance ceases to be described when it is traced back to the force of "will," or to the property of "excitability." The fault of which we are most often guilty, even when we have fully recognised the distinction between comprehensible relation and incomprehensible essence, is to *prematurely* invoke a final cause. The fault of which we are sometimes guilty is, after having invoked a final cause, *a quo* or *ad quem*, whether prematurely or not, to proceed through the dead end as if the phenomenal sequence were unbroken. And still grosser faults are possible—faults of observation, faults of interpretation, faults of language, and faults of candour.

¹ What consciousness is we do not know. All that we do know is consciousness. Obviously the paradox depends upon thimble-rigging with the word "know."

The particular theme now submitted to analysis is the functional significance of the cerebral cortex; the question to be answered may be put thus: Have we reached any true dead end to knowledge in the conclusion that the cerebral cortex contains "sensory centres" and "motor centres," and if so, what signification do we attach to these final terms?

It will, I think, be of service towards the answer to these questions to begin by quoting some of the most pregnant passages from Hitzig and Munk. Owing to the wealth of the German language as regards psychological terminology, in comparison with English or French, the German interpretation has been by far the most searching, and by reason of this very wealth as well as by reason of the subtle harmonics and connotations in the individual words, that interpretation has remained practically ignored by us. Even Ferrier has not in his "Functions of the Brain," given at all an adequate account of Hitzig's view, still less of Munk's; Bastian ignores them both; and James, while giving abundant evidence of our great indebtedness to Munk, strangely enough goes the length of saying "that Munk's absolute tone about his observations and his theoretic arrogance have led to his ruin as an authority."

The most weighty critic of Munk is Goltz, less on account of his polemical skill than because of the weight of his experiments. We cannot but hesitate from an unreserved admission of Munk's data in so far as they have been derived from dogs, when Goltz brings before us dogs, first with one hemisphere removed, then with both hemispheres removed, apathetic and stupid it is true, yet able to feel, to move, to snarl and bark, to stand on their hind legs, to see and even to feed, and liable to have epileptic fits.¹

¹ GOLTZ. Siebente Abhandlung über die Verrichtungen des Grosshirns. *Pflüger's Archiv.*, li. 1892, p. 570.

Wundt has recently published a critical review of Munk's doctrine of cortical projection in the 6th volume of his "Philosophische Studien," 1890. His criticism is particularly directed to Munk's retino-cortical scheme as being inclusive of two non-congruent conceptions of centralisation, relating (1) to distinctness of vision (sensation) (2) to complication of vision (perception). He characterises the view that high-level vision, hearing &c., are effected at distinct foci of visual, auditory, &c., areas, as a revival of the phrenological faculty theory, and as leading into a psychologically untenable position.

But the question of interpretation may to a large extent be kept distinct from that of localisation; certain principles if true, are true whether cortical functions be localised or concentrated or diffused.

I have already on a previous occasion¹ pointed out the identity of idea between Munk's Seelen-blindheit and-taubheit, Rinden-blindheit and-taubheit, and Hughlings Jackson's well-known hierarchic scheme. And in translating Munk's language I shall take the liberty of rendering the German prefixes Seelen—and Rinden—by the English prefixes higher-level and lower-level, a rendering which appears to me to be less question-begging than a literal translation of these two very heterologous words, and justified by what Munk himself says of the sense in which he uses the terms.²

Hitzig³ says :

"A portion of the convexity of the dog's cerebrum is motor (using this expression in Schiff's sense), another portion is not motor."

"The motor portion, speaking generally, is situated anteriorly, the non-motor portion posteriorly."

"By electrical excitation of the motor portion one obtains combined muscular contractions of the opposite half of the body."

"With very weak currents the muscular contractions can be localised in definite and narrowly restricted groups of muscles. With stronger currents excitation of the same or of closely adjacent spots, causes other muscles, and even muscles of the same side of the body to participate in the contraction. The possibility of isolated excitation of a definite muscular group,

¹ "An Introduction to Human Physiology," 1891, p. 533.

² "Ich habe diese Bezeichnung im Jahre 1877 nach vieler Ueberlegung gewählt und ihr vor "Vorstellungsblindheit" oder "Erinnerungsblindheit," die am nächsten lagen, aus guten Gründen den Vorzug geben zu müssen geglaubt. Indem ich wiederholt definirte: Seelenblindheit=Fehlen der Gesichtsvorstellungen, der Erinnerungsbilder der Gesichtswahrnehmungen, dürfte ich die Benützung von "Seele" für gerade so unbedenklich halten, wie wenn ich α Blindheit oder β Blindheit gesagt hätte. Dass trotzdem Missverständnisse von seiten flüchtiger Leser nicht ausgeblieben sind, kann mich um so weniger veranlassen die Bezeichnung aufzugeben, als ich eine bessere noch bis heute nicht gefunden habe." Munk, Gesammelte Mittheilungen, 2nd edition, note to p. 41.

³ Hitzig. Untersuchungen über das Gehirn. Berlin, 1874, p. 11.

Fritsch and Hitzig. du Bois-Reymond's Archiv., 1870, p. 310.

using very weak currents, is, however, limited to very small spots, which for the sake of brevity, we may designate as centres."

"It is an assured fact that a considerable portion of the nerve-mass forming the cerebral hemisphere (say nearly one-half) is in immediate relation with muscular movement, and that another portion, at least directly, has no evident connection with it."¹

"If we raise the question whether our excitation effects were caused by a direct action upon particular centres in which the voluntary motor impulse arises, or whether they are attributable to an excitation of subjacent fibres, or whether any third possibility exists, our answer must be far more reserved."

"Even if we assume as proved, that the movements in question are liberated by the ganglionic substance—and it is not proved—this would not be enough to prove that with these movements which are liberated by the internal event, that precise portion of the cortex furnishes the substratum of the first outward step in the series beginning with the origination of a sense-impression, and finding a temporary end in the expression of will evidenced as muscular movement."

"It is, on the other hand, far from inconceivable—and the notion is favoured by our knowledge of the anastomotic structure of these parts—that that portion of the brain which includes the birthplace of a will to move, is of another, or perhaps of a more complex nature, and that the parts which we have called centres only constitute agencies, exchanges, in which an arrangement of muscular movements occurs, similar to that effected through the grey matter of the spinal cord and basal ganglia, but more purposeful."

And later (page 30) describing the state of the two dogs upon which circumscribed portions of the brain (the "centres" for the anterior extremity) had been extirpated.

"Both animals, after extirpation of a portion of the (by us) so-called centre for the anterior extremity, had only partially lost the power of moving that limb, and had apparently suffered no loss of sensibility. But they evidently had only an imperfect knowledge of the state of this limb; they had lost the power of forming to themselves complete representations of its states; they were thus the subjects of a symptom, which appears

¹ Untersuchungen, p. 25-26.

in a very similar fashion in certain manifestations of tabes, only in this case, there was certainly no lesion of a sensory tract. One might express himself thus to characterise this condition more closely. Some kind of motor communication still persisted from mind to muscle, whereas, somewhere in the connection from muscle to mind there was a break. Possibly this break affected the terminus of the hypothetical path of the muscular sense, in any case it coincided with the position of the injured centre."

"Speculations concerning cerebral and mental activities have deservedly fallen into such discredit, that one may wisely observe an excess of caution in dealing with such matters."¹

". . . We have, hitherto, avoided committing ourselves to a definite opinion whether we excited cells or fibres, termini or stations in the psychomotor circuit, and we shall still refrain from doing so. On the other hand, we have proved that the stimulus did act through known reflex channels. But if anyone should conclude, on account of these differences [polar differences, length of contraction] that the parts originally excited were not central expansions of the motor nerves, he clearly would have no grounds for the conclusion. For there is nothing to prove that central fibres (or cells) in connection with peripheral fibres (or cells) must react precisely like these peripheral elements."

"On the contrary, it is probable from all that we know that they react differently. And finally, the effects we have described have no analogy whatever among peripheral phenomena. One must, indeed, rest content with the assumption that the peculiarities depend upon the peculiar properties of the central organ."

". . . And yet I regret to have used the expression 'muscular sense,' for it has caused all kinds of misunderstanding from the very first. Nothnagel has, meanwhile, characterised the symptoms in question as disturbances of 'muscular sense,' far more definitely than I have. This determines me to specify my own conception more precisely, although I am most unwilling to depart from the reserve that I have imposed upon myself. For dealing with these factors as they become better and better known to us, an unknown magnitude and one which can hardly be ignored forces itself upon us—the Will. We stand towards this factor, as we do towards the elementary forces; we may know the modes of its manifestation, but not its essence, nor its actual nature."

¹ Untersuchungen, p. 52-53.

"We characterised the state of the animals we had operated upon as follows: They evidently had only an imperfect knowledge of the state of the affected limb; they had lost the power of forming complete representations of it; and we were justified in this conclusion by the analysis of the perturbations of movements that existed after destruction of that spot of the cortex, the excitation of which had caused movements of the now abnormally acting muscles. The question now before us may be thus simply formulated."

"Is the excluded centre the organ which alone governs the movements in question, or is there in addition to the general organ of will—assuming such to exist—another central motor organ in the same circuit of discharge?"

"To be as brief as possible we may refer to common experience of moderate movements by normal muscle. It is known that the image of movement caused in our own consciousness by muscular action is exceedingly clear. An expert painter would for instance be able to faithfully reproduce a posture assumed by himself with closed eyes. Nevertheless consciousness knows nothing of the moving factors; even the representations obtained by direct observation or otherwise, are so intimately connected with another department, that they contribute but little to the perception of motor forces regularly operating at the periphery. These are indeed scarcely known to us as regards each particular form of movement, they are only discoverable by inference; and digital palpation, *e.g.*, is far more helpful to this end than the repetition of the movement."

"Still it is clear that very exact representations of the state of muscle must be produced—as we learn from these very images of movement—and it is equally clear that these images are attributable chiefly to the perception of the muscular state, and only in minor degree to joints, skin, &c.: this we learn from the well known illusions of movements occurring in the paralysis of ocular muscles."

"If nevertheless our representations of the muscular state of our own body do not overstep the threshold of distinct consciousness, and thus enable us to see into the true nature of the process, we must attribute this failure to a very general law. We are able to distinguish the state of particular organs only in so far as is necessary and sufficient for their use in the uninterrupted maintenance of their function."

"But within such limits, the apprehension of these mainly unconscious representations of each particular phase of move-

ment, constitutes one of the necessary conditions of a normal progress of its succeeding phase; subsequently, (considering apparent muscular repose as a phase of movement) one must recognise that muscular states in general are among the various causes that guide the organism in its voluntary movements, and that regulate these movements. Let us assume an entire absence of all other sensory stimuli and perceptions, so that we have to do with a simple motor machine of such a kind set in motion by the muscular impulse, we may then very well imagine it as sufficient for the execution of purposive movements."

"We have recognised in the above described portions of cortex an organ the function of which coincides with that aspect of the psychical phenomenon that we have been considering, and I do not see the necessity for admitting that Will as such involves a specific and different motor organ. When in consequence of the joint action of several sensory impressions, either present or past, the motive of a movement arises, such motive never takes form thus: "Innervate muscle a b c, so that arm n, shall move through angle x," but thus: "take," "write," "speak," &c. The organs we are acquainted with, appear to me sufficient to enable us to form a conception of the normal progress of the movement when once it has been thus initiated. Points of detail are it is true still sufficiently obscure." (*Untersuchungen*, p. 59-61).

Munk¹ in his third communication to the Berlin Physiological Society says:—

"I have now to direct your attention to a third department of the cerebral cortex of the dog—the portion CDE, Fig. 1. You recognise at once that it is the portion of the cortex upon which so much consideration has been bestowed during the last eight years, since the first investigations of Fritsch and Hitzig. If, nevertheless, a clear understanding has not been arrived at as regards the functions of this cortical department, this must be attributed to the fact that we do not yet possess a guiding point of view for a correct appreciation of the experimental data. The further I advanced in the knowledge of the auditory, and more particularly of the visual region, the more orderly became data that appeared at first irreconcilable; and if last year I was obliged to content myself with presenting to you our cortical department as the motor area in contrast with the posterior sensory area, now I believe myself able to invite you to a deeper comprehension of this cortical region as well."

¹ MUNK. *Du Bois-Reymond's Archiv*, 1878, p. 162.

"In a word our cortical region CDE is the sensory area (Fühlsphäre) of the dog. Just as the cortex of the occipital lobes stands in relation to vision, and that of the temporal lobes to hearing, so the cortex of the parietal area stands in relation to common sensation (Gefühlsinn); in this as in the other cases we have the locus where perception is consummated and where representations, the memory images of perceptions, have their seat. Let it be clearly understood, however, that it is not sensation of the skin only that is here in question, but sensation in its broadest sense, the sensation of the whole body." (p. 171.)

". . . After extirpation on a dog of a portion of cortex in the area CDE of one hemisphere, the sole and regular consequence (when inflammatory reaction has passed off) is disturbance of motility and sensibility on the opposite half of the body, in one part or in another according to the situation of the extirpated spot. With the lapse of time these alterations gradually vanish, sometimes less, sometimes more completely, so that finally in the most favourable cases our dog recovers the demeanour of a normal dog."

". . . Still these disturbances are very imperfectly characterised by the terms, disturbance of motility, disturbance of sensibility." (p. 173).

Then follows a careful description of the state of matters in a representative dog having suffered extirpation of D on left side, three to eight days after operation.

"The cortex of the parietal lobes of the dog is the sensory area of the opposite half of the body, and is composed of a number of regions each of which is related to a particular part of this half. In the percipient central elements of each region terminate contiguous fibres that subserve cutaneous, muscular and neural sensations (Innervationsgefühle) of each corresponding part of the body. Within each such region the sense-representations (Gefühlsvorstellungen) of each such part of the body have their seat, so that the former constitute the special sensory areae of the latter, *e.g.*, of the anterior or of the posterior extremities. Within the province of each such sensory area of particular parts of the body, small extirpations entail partial loss of sensory representation of the part, larger extirpations its complete loss—high-level paralysis (loss of high-level motility and of high-level sensibility) of the part; but in any remaining parts of a sensory area there can be a restoration of sense-representation.

By still more extensive extirpation, low-level sensations may be impaired, and only a portion of high-level sensation may be recoverable. The impairment and the failure of restitution are the greater, the smaller the remaining sensory area. Its complete destruction on one side has as its necessary consequence permanent loss of all sensation—low-level as well as high-level—total paralysis (total loss of movement and of sensation) of the related part of the body." (p. 176.)

" . . . Perceptions and representations in the domain of common sensation (Gefühlsinn) have been so variously characterised and defined by various authorities, that in the absence of a particularly solid groundwork, our study of the sensory area would have been completely fruitless. I have, therefore, prefaced our consideration of the sensory area by a survey of sensory perception, by going over the organs of the body, the nerves of which subserve perception, and from which perceptions evolve representations. Still, in the consideration of neural sensations, I have restricted myself to defining them as perceptions derived from motor impulses accompanying active movements; it thus remains to be added what are the organs, of which the modifications can reach consciousness as neural sensations. These organs, as I have stated above, are the subcortical ganglia or centres in brain and cord, that invoke movements. As in the infancy of the animal, the representations of movement are developed from its first purely reflex movements; as in the adult animal, the representations of movements of a part of the body, can still arise in its sensory area, even if (*e.g.*, in locomotion), this sensory area is not actually participating in the accomplishment of such movements; as finally, the representations of movement lost in consequence of cortical extirpation in a given part of the sensory area, may be reformed out of the reflex and automatic movements of the affected part, it cannot but be that fibres ascend to the cerebral cortex from subcortical motor centres or ganglia, as well as from the skin and from the muscles—fibres that subserve cortical perception of these subcortical centres."

"Our neural sensations are indeed, for the present, to be distinguished from what has hitherto very generally received the name of neural sensation (Innervationsgefühl)—from the "perception of the intensity of voluntary effort in connection with voluntary movement. "Will," "voluntary movement," with seat and origin in the cerebral cortex, are indeed very convenient, and may, therefore, also be valid expressions, but they are destitute

of a phenomenal physiological substratum. All we know of the cerebral cortex is that it is the place of perceptions and the seat of representations. Beyond this, it is merely admissible to assume as has been assumed by Meynert on somewhat different data, and by Wernicke, that representations of movement are the causes of so-called voluntary movements; that with the production of such a representation to a given degree (and indeed with a production *viâ* association, not *viâ* its ordinary constituent sensations) *eo ipso* the corresponding movement is elicited; and that the greater its formative representation the greater the resultant movement. The "perception of the intensity of voluntary effort, in connection with voluntary movement," might indeed be the attribute of a representation of movement; a true perception might still obtain only indirectly, quite independently of the "Will," and the percept would then be nothing but the neural sensation in the sense just set forth." (p. 177-8.)

§2. SENSORY AND MOTOR CENTRES.

"Sensory" and "Motor" are used almost antithetically. "Sensory" connotes *physically* centripetal changes ending *subjectively* in feeling. "Motor" connotes *physically* centrifugal changes beginning, according to some thinkers spontaneously in a motor centre, according to others consequently upon previous centripetal changes.

I need hardly say that I adopt the second view, every centre must be sensori-motor, *terminus ad quem* as well as *terminus a quo*; and in the two different components of the double term I do not recognise any phenomenal division of the central process into sensory and motor. Strictly I picture one undivided change taking place in any centre, resulting from impulses to it and resulting in impulses from it; therefore as far as possible I avoid the term motor, believing that it is apt to imply the spontaneous origination¹ of force, but if speaking loosely in accordance with custom, I occasionally use this term and refer *e.g.*, to a motor area, I feel it impossible not to add a guarding phrase to exclude that crude and (to me) inconceivable implication. Between centripetal and centrifugal impulses, I see a single physical process, one.

¹ This is explicitly declared in Bain's theory of Spontaneity.

and indivisible; to call it sensory or to call it motor, or even to call it sensori-motor, are to my thinking, imperfectly and improperly to designate it by more or less subjective terms—with more or less obstructive connotations. All that I can recognise in the notion of a centre is an organ of elaboration receiving and giving out impulses. By the term motor I denote that it emits but fails to denote that it receives; by the term sensory I denote that it receives (and imply that *It* feels) and fail to denote that it emits impulses; by the term senso-motor I denote reception, “feeling,” and emission. All these meanings when closely analysed are illegitimate, and convey too little or too much. Experimentally I may not predicate “feeling” of any centre, but only of the hypothetical *ego*, I may only infer from visible movement that other animals “feel” and that sensations similar to my own are associated with the activity of certain centres.

This inference is unavoidably included in the word “sensory” every time we use it, and it is only in the loose language of the laboratory that we have any justification for speaking of a “sensory centre.” We are always in presence of a process manifesting itself by movement or by want of movement, and although we may for practical purposes say in the laboratory that such a centre is sensory because after its removal signs of sensation have vanished, or motor because after its removal paresis has resulted, we have no strict logical right to say that a centre is sensory, or that it is motor, nor even that it is sensory and motor. We are in presence of a central process associated with centripetal and centrifugal processes, and we have no right to say that the centre is motor, or sensory, or both, otherwise than inferentially, and guarded by a clause of interpretation to indicate that we are conscious of the inference included in the term. The inferences contained in these qualifying adjectives are at once recognised by placing oneself in the critic’s attitude. How do you know that the central process is motor and not sensory? Answer—By the movements which are its consequence, and by the absence of movements which I have learned to be expressive of sensation. How do you know

that the central process is sensory and not motor? By the movements which are its consequence or by the absence of movements which are the consequence of its annihilation, and here we clearly see that the process if sensory must also be motor, directly or indirectly, if it is to manifest objective signs. How do you know that it is sensori-motor? Motor by its movements, sensory of necessity. But in this answer we finally see that if motor may be predicated of a process seeing that it is the antecedent of motion, the word does not predicate enough, while to add "sensory" predicates too much.

So that in sum we may say—the central process is motor, *i.e.*, it produces movement; it must have centripetal antecedents, *i.e.*, motion is not spontaneously generated; it may be sensificatory, *i.e.*, a conclusion inferred from the data *sensatio mea et motus tuus*. "Sense sure you have, else could you not have motion," and we may find it convenient to use the term motivity or senso-motivity to designate this property of certain nerve-centres.

Is it not plain that inference from experiments as to motor and sensory centres and processes must have much deceptive verbal connotation to very little phenomenal denotation? What are the phenomena denoted by the "sensory centre" of a dog or the "motor centre" of a monkey, by a sensation, by an "afferent centre"? And how slight have often been the phenomenal data from which it has been said that this animal has lost such and such a sensation, that such and such a function is *localised* in this or that part of the brain. I understand the phenomenal meaning of an afferent tract or of an efferent tract, but not that of a motor centre or a sensory centre. I picture a wave of change passing through a cell, but do not know at what transverse section of the wave to label it motor or sensory. The property of cortical grey matter is sensomotivity; the most typical so-called motor cortex is senso-motor, the most typical so-called sensory cortex is senso-motor.

This has been no merely verbal disquisition, but on the contrary is fundamental to the subject. We must recognise that one indivisible *thing* is cut into two ideas by these *words*

“sensory” and “motor.” A central process is not sensory or motor but senso-motor (in a guarded sense), and a centre is an “organ of return of action.”

Let us apply this notion to the central process which we consider as associated with muscular movement, and see by its aid what we are to understand by such expressions as “motor idea,” “idea of movement,” “kinæsthetic idea.”

By those who make a distinction and contrast between a motor and a sensory process, the motor idea is a mental picture of *movements about to be made*, the kinæsthetic idea is a mental picture of the results of *movements just made*. The motor idea is considered as an antecedent to motion, the sensory idea as its consequent. It does not matter whether we further admit that we may have the idea of movement without actual movement, or as the antecedent of a future but deferred movement, or as the consequent of a movement made long ago, we must in all cases recognise that it is illegitimate to say that the idea of movement is exclusively a motor idea, an image of an imminent motor discharge, or that it is exclusively a sensory, *i.e.*, kinæsthetic idea, an image of the results of previous similar movements. It is both. Each movement is an element in a series of movements, its idea or image is at once the consequence of past movements and the antecedent of the movement in consummation, which movement in consummation as fast as it progresses is attended with renewed impressions from the periphery pouring into the sum of past sensation at the centre. In a movement made at this moment, the image of the past similar movements is at the same time the image of the actual movement, which by its results repeats a similar image guiding the movement itself in its progress, and educating the centre towards future similar movements. It is not possible to distinguish a “kinæsthetic” image of past movements from a “motor” image of impending movements. The two words denote one thing.¹

¹ Thought of movement is memory of movement, or more generally the *thought* is the *remembered*. An impulse, an intention, a resolution, a prayer, is a more and more concentrated act of attention, of memory, of thought. An identical neural process is the essential phenomenon wrapped up and presented in these very different words, will, attention, memory, belief, thought.

Nevertheless we can and do recognise in the use of the word *sensio-motor*, which connotes the notion that the centripetal generates the centrifugal phenomenon, the principle that phenomena have generators and consequents antecedents; we are reminded by the word that a centre is an organ of return of action and that the type of all motor action is a reflex act. This principle is recognised by all leading workers and thinkers—by Hitzig in his conception that the motor area of the cortex is a “muscular sense” area; by Munk in his conception that it is the motor “Fühlsphäre;” by Bastian in his conception that it is the centre of “kinæsthetic” impressions. These three conceptions are but one and the same conception, which I most explicitly and unreservedly accept as a fundamental article of thought.

But beyond this fundamental and simple article that all action is literally “ex-cited,” that any centre is an organ of return of action, or more generally still, that as far as our experience goes all phenomena are generated, not self-created, the ways part, and in the further description of the neural government of the body, which various observers and thinkers feel themselves entitled to give, variety and contradiction arise. Yet neither a purely motor view on the one hand, nor a purely sensory view on the other, can constitute for us a complete and satisfactory theory; each view is true in so far as it contains half the truth, but neither is true in so far as each contains only half the truth; the whole truth is contained in the sum of both views. The Rolandic area is *sensio-motor*. And in the mental picture that we form of the neural government of the body, it is not sufficient to admit by itself, either the undoubtedly true principle that all motion immediately or remotely is an outcome of centripetal constitution, or the equally undoubted truth that the cortex is “motor” in so far as its excitation immediately or ultimately produces motion—but we must also admit (as I have urged elsewhere) that “in the co-ordinate function of any synergic group of muscles at least three conditions must be fulfilled: (1) impulses must be emitted from the centre in appropriate manner and degree; (2) the muscles must be ‘in touch’ with the spinal

axis, duly responsive to the reflex ebb and flow arising from altering peripheral states; (3) the muscles must be in touch with each other, directly reacting against that passive extension which antagonist muscles undergo from each other."¹

It will however be convenient to postpone further consideration of this view to a later stage.

§ 3.—CORTICAL CENTRES.

A little reflection and a very slight acquaintance with "la belle doctrine" of cortical localisation, as that doctrine has been stated and supported and controverted by various authorities and writers, will very adequately correct any disposition we may feel to regard the examination of the terms used as a barren dialectical exercise. The differences of view have involved matters of fact far less than inferences from fact as embodied in words; identical words having been used of very unequal phenomenal value, and similar phenomena having been dressed up in words of apparently widely differing and even contradictory meaning. The effects of excitation and of lesion in the Rolandic area evidently have not been and cannot have been of such different phenomenal value under the hands of Fritsch and Hitzig, Ferrier, Munk and Goltz, as seems to be indicated by the terms "muscular sense centre," "Fühlsphäre," "motor centre," "sensory centre."² It can only have been by a defect in the valuation of words that we have had presented to us denial of localisation, in presence of descriptions of "motor" and "sensory" centres as definite as descriptions of carrots and of turnips.

The form which the verbal controversy has taken in this country is represented with greatest clearness and

¹ WALLER. "Physiology," p. 466, also pp. 341-2.

² I need hardly point out again that my paper does not directly involve any question of actual localisation. Its main arguments are equally applicable, whether "sensomotility" be the property of the entire cortex or of only its Rolandic area, whether we admit motor and sensory centres as taught by Hitzig and by Ferrier, or the cortical map of Munk, or indifference of function in the unrestricted sense of Flourens, or in the restricted sense of Goltz.

intensity by the Ferrier *v.* Bastian issue concerning the functional significance of the Rolandic area.

In the early days of cortical experimentation Ferrier designated the results of cortical stimuli as "motor." He says:¹

"By the term motor centre, as applied to a particular part of the cortex of the brain, I have endeavoured to signify the fact that this part is in direct communication with the motor tracts and their ganglia, and that its function is to excite co-ordinated muscular action of a definite kind of the nature which we call voluntary."

Here and elsewhere Ferrier defines the sense in which he uses the term "motor," and having done this is legitimately entitled to use the term to designate as "motor," those parts of the nervous system which he finds to be concerned in the production of movements.

But Bastian, confining his attention to Ferrier's experiments, altogether objects to the word "motor," emphatically denies that the Rolandic area is motor, and designates it as a "Kinæsthetic" area. It is not difficult to see how this apparently fundamental difference has arisen. Ferrier had in view movement and its central origin, and used the term motor as if implying the spontaneous origination of motor impulses in supreme psychical centres; he nowhere defends himself from the truism that phenomena cannot, as far as we know, be spontaneously generated, and that what we call a "motor" impulse is necessarily not an uncaused motor impulse, but a resultant of antecedent phenomena; indeed he appeared to adhere to a view of central initiation by the introduction of the somewhat unfortunate term "psychomotor." He may or he may not, consciously or unconsciously, have been influenced by the well-known Aberdeen doctrine of motor spontaneity as taught by Bain. In so far as Bastian entered a *caveat* against this neat "motor" doctrine at a time when it was dominant in this country, he rendered good service to our true knowledge of the subject. And that service would have been still more complete if it could have been made plain to us that kinæ-

¹ West Riding Lunatic Asylum Reports, 1874, p. 47.

thesis implied neither more nor less than is expressed in the analyses of Meynert, Wernicke, Schiff, Fritsch, Hitzig, and Munk. Then the turn might have been understood and welcomed as the expression of a familiar German postulate, viz., one indivisible process—kinæsthesia, ideally composed of æsthesia leading to kinæsis. If that is the sense in which Bastian has used the term, I unreservedly accept it as denoting a fundamental postulate.

The phenomena antecedent to "motor impulses," (leaving on one side effects of nutrition or injury or experimental stimuli), are what are commonly designated as sensory, or more properly centripetal phenomena. Whereas Ferrier with his attention focussed upon the effect, calls the centre to which he traces it "motor," Bastian goes back a step further and refers the muscular movement to its centripetal antecedents which he designates "kinæsthetic." These antecedents are for him so striking that he denies their central resultant to be motor; yet he recognises that centrifugal processes occur along efferent tracts (which he designates "internuntial") connected with the Rolandic area, and it is plain that in denying that that area is "motor" in Ferrier's sense, all that he really denies is that it is *spontaneously motor*. He contradicts "la doctrine brutale" of motor centres.

If anyone can doubt that this is the correct interpretation of Dr. Bastian's position concerning the Rolandic area, his mind will be set at rest by referring to the diagram, in which Dr. Bastian has represented the physiological anatomy of the "kinæsthetic" process. (BRAIN, 1892, p. 31. Fig. 1.)

He has there made the matter perfectly clear, and in so doing has shewn how unnecessary it has been to burden and obscure his presentation of so simple a matter by such terms as "kinæsthesia" and "internuntial." The facts presented are admitted by everybody, the words in which they have been clothed are mystificatory. And the principal part in this disguise is evidently that played by the word "internuntial" as applied to the pyramidal tract. Dr. Bastian's contention is that the pyramidal tract is not efferent but internuntial, that therefore the cortical area from which

it proceeds is not motor. In Dr. Bastian's diagram a Rolandic centre is pictured in connection with an afferent tract and an efferent (internuntial) tract, a spinal centre on the afferent tract is designated afferent, that on the efferent tract is designated efferent; but the Rolandic centre (by calling the pyramidal tract internuntial and not efferent) is made to be on an afferent tract only. Therefore the Rolandic centre is not "motor," the only "motor" centres are bulbo-spinal.¹

That Dr. Bastian's views are principally if not exclusively determined in opposition to the views of Dr. Ferrier, is equally clearly indicated in this his most recent publication.

"The so-called motor centres of the cortex were not, of course, originally supposed to be termini for afferent impres-

¹ It is surely inconvenient to speak of afferent and efferent centres, afferent and efferent impulses, and more verbally correct to speak of afferent and efferent tracts, centripetal and centrifugal processes. As regards centres the difficulty of qualification is greater. We may indeed speak of afferent and efferent centres when they are known to be on afferent and efferent tracts; but as regards cortical centres this cannot be done. "Motor," "Sensory," and "Sensori-motor" are the words which I find it necessary to use in elementary teaching, but they are not unobjectionable. The central terminus is *ad quem* and *a quo*; but whether the arrival and departure platforms are conjoined in one "Sensori-motor" centre or in "sensory" and "motor" centres commissurally joined, is a different question. The present tendency is to accept the "sensori-motor" character of the cortex in the sense indicated by Munk's account of his own experiments.

The truism that by sensory centre or motor centre we cannot mean a centre which feels or which causes motion, but at most a place where sensation occurs or a motor impulse is elaborated, is worth making particularly clear if only as a matter of definition.

I have two laboratories, in one of which I learn, while in the other I teach. The first is my sensory laboratory, the second is my motor laboratory, and the Edgware Road is the main internuntial tract between them. But the laboratories are themselves neither sensory nor motor, they are places in which sensation and motion are produced. And in this restricted sense they are not purely sensory, nor purely motor; the first is motor through the second, the second is sensory from the first.

One of my friends has a single large laboratory in which he learns and teaches. It is his senso-motor laboratory, and as a matter of convenience it is divided into rooms in which most sensation occurs, and rooms in which most motion occurs. But here again the rooms are not purely sensory nor purely motor.

Now *a priori* it can be that in that psycho-physical laboratory called the cortex cerebri there are sensory departments and motor departments. It cannot be that the cortical departments are purely sensory or purely motor, but to what degree, if any, sensation and motion and particular forms of either are concentrated or localised in particular departments can only be ascertained by the *a posteriori* method, by actual experiment.

But experiments are conflicting, Goltz's dogs absolutely contradict the dogs of Munk, and complete (*i.e.* logically insulated) data on the human brain are few.

"sions ; when first discovered, they were said and they are still "maintained by Ferrier to be true "motor centres." (BRAIN, "1892, p. 30).

As a matter of fact, the excitable area of the dog's brain when first discovered by Fritsch and Hitzig, was by them inferred to be a "muscular sense area." Ferrier, while maintaining that the Rolandic area is "motor" inasmuch as it gives rise to motion, most assuredly would not assert them to be "true motor centres" in the absolute and exclusive sense implied by Bastian.

Goltz in his first¹ (and subsequent) papers on the brain, lays far more stress upon loss of sensation than upon loss of motion in consequence of cortical ablation.

Schiff² interpreted Hitzig's extirpation and excitation effects to signify that the cortex is organ of return, and lays stress on the loss of sensation due to extirpation.

The necessary functional connection between "motor" cortex and centripetal impressions (using "motor" in its carefully guarded sense to qualify a point of cortical departure and not a point of origination), is experimentally demonstrated in the observations of Bubnoff and Heidenhain.³ Cortical excitations (dog) producing contractions after an interval of .03" to .04", did so during excitation of the central end of the divided sciatic⁴ after an interval of .6" to .12". Here was a retardation of "motor" elaboration, due to centripetal impressions ; under different conditions a precisely opposite effect of the same cause was observed, viz., a shortening of the reaction-time, *i.e.*, a sign of increased "motor" or centrifugal tension.

And the whole spirit and tendency of Munk's investigations have been in the broader sense which is thus entirely ignored by Dr. Bastian. From first to last (1877 to 1889) Munk, in his interpretation of his own results, holds prominently before us the "arrival aspect" rather than

¹ Goltz. *Pflüger's Archiv*. 1876, xiii., p. 9.

² Schiff. *Rivista Sperimentale*, 1876.

³ *Pflüger's Archiv*. xxvi., p. 164, 1881.

⁴ It is, perhaps, not superfluous to say that this is not quoted in support of any centripetal influence along an efferent tract.—*Vide infra*, p. 382 *et seq.*, and Bastian (BRAIN, 1892, p. 26, footnote.)

the "departure aspect" of cortical processes, so prominently indeed that he has given to the Rolandic department of the cortex the name of Fühlsphäre.

Professor James interpreting the physiological phenomena from a psychological and critical standpoint, comes to a very similar conclusion; he very happily compares the motor area to the "mouth of the funnel," through which pour outgoing impulses, caused by incoming impulses. His analysis also leads him to recognise the illusory character of a sharp distinction between motor and sensory centres.

"... so that really it (*i.e.*, a 'motor' centre) is only the "mouth of the funnel, as it were, through which the stream "of innervation, starting from elsewhere, pours; consciousness "accompanying the stream, and being mainly of things seen "if the stream is strongest occipitally, of things heard if it is "strongest temporally, of things felt, &c., if the stream occupies "most intensely the motor zone."—(*Psychology*, vol. i., p. 65.)

I very cordially agree with this kind of verbal representation, distinctly adding, however, that I do not agree with the arbitrary and dogmatic exclusion of the motor zone from the "sphere of consciousness," elsewhere enunciated by Prof. James, and approvingly quoted by Bastian, Ferrier, Gotch, and Horsley.

"If the motor cells are distinct structures, they are as "insentient as the motor nerve-trunks are after the posterior "roots are cut. If they are not distinct structures, but are "only the last sensory cells, those at 'the mouth of the funnel,' "then their consciousness is that of kinæsthetic ideas and sensations merely, *and this consciousness accompanies the rise of "activity in them rather than its discharge.*"—(*Psych.* ii., p. 517.)

It is to the last sentence mainly, indicated by italics (not in original¹) that I take exception. *Non sequitur.* The assertion is no more than an assertion; it is no necessary associate of James' second alternative, and, in my view, he has no right to thus distinguish between the "rise" and the "discharge," nor to attribute consciousness exclusively to either.

¹ In all other parts of this paper italics in quoted passages are italics in the originals.

"All paths are paths of discharge, and the discharge always takes place in the direction of least resistance, whether the cell which discharges be 'motor' or 'sensory.' The connate paths of least resistance are the paths of instinctive reaction; and I submit as my first hypothesis that *these paths all run one way, that is, from 'sensory' cells into 'motor' cells, and from motor cells into muscles, without ever taking the reverse direction.*

"A motor cell, for example, never awakens a sensory cell directly, but only through the incoming current caused by the bodily movements to which its discharge gives rise. And a sensory cell *always* discharges or normally tends to discharge towards the motor region. Let this direction be the 'forward' direction. I call the law an hypothesis, but really it is an indubitable truth. No impression or idea of eye, ear, or skin comes to us without occasioning a movement, even though the movement be no more than the accommodation of the sense-organ; and all our trains of sensation and sensational imagery have their terms alternated and interpenetrated with motor processes, of most of which we are practically unconscious. Another way of stating the rule is to say that, primarily or connately, all currents through the brain run towards the Rolandic region, and that there they run out, and never return upon themselves. From this point of view, the distinction of sensory and motor cells has no fundamental significance. All cells are motor; we simply call those of the Rolandic region, those nearest the mouth of the funnel, the motor cells par excellence."—(*James' Psychology*, vol. ii., p. 581).

I am again in agreement with this mode of view, and most unreservedly so with the concluding paragraph. But I would appeal to Prof. James himself, to bear witness to the inconvenience of thought and expression into which he must have repeatedly found himself forced by the use of these two words, "motor" and "sensory," with their implications of distinction between motor centres and sensory centres. I have elsewhere given expression to the same idea in the following terms:

"Any motor or discharging centre must also be a 'sensory' or receiving centre; it must be excited as well as excite. Any 'sensory' centre must also be motor, directly or indirectly; else we could have no objective tokens of sensation; every centre, whether called motor or sensory, is *terminus ad quem* as well as *terminus a quo*."

And with regard to the law of forward direction I fully agree with James that it is not an hypothesis but an indubitable truth, at any rate as regards efferent channels. That the vortical movement, of which organisms are the scene, and their nervous systems the highest organic expression, *should* exist in one direction is *a priori* probable. That the vortical movement *does* normally exist in one direction in the nervous system, is known to everyone who knows that stimulation of the central end of an afferent nerve, or of the peripheral end of an efferent nerve produces movement, while stimulation of the peripheral end of an afferent nerve, or of the central end of an efferent nerve produces no movement. Yet that it *may* possibly be produced in an opposite direction, at least in centripetal channels, is indicated by more or less convincing considerations, viz., by Du Bois-Reymond's negative variation in the posterior roots, and by Gotch and Horsley's observations of spinal discharge, *viâ* the posterior roots. Wundt indeed accepts the possibility to such an extent that he speaks of "sensory-centrifugal" channels and accepts without comment the view that a hallucinatory disturbance of the brain may propagate itself down the optic nerve to the retina.¹ But to weigh this doubtful point is foreign to my present purpose.

That an effective impulse cannot occur *up* an efferent channel, is proved by the absence of reflex action when an efferent nerve is excited.²

§ 4—ELEMENTARY CONSIDERATIONS.

I had fallen into the habit of imagining neural processes on a three-level scheme, before even knowing the name of Hughlings Jackson, and had adopted as typical diagrams of reference the two schemata (Figs, 1 and 2) which I now reproduce from notes put on paper in 1876, adding to them the expressions then used, and the Jacksonian expressions that I have since learned. But I have never regarded such a three-level scheme as anything more than a convenient

¹ *Physiol. Psychol.*, vol. ii., p. 432 (3rd. ed.) Engelmann's retino-motor fibres are not here in question.

² See also page 386.

scaffolding upon which to hang certain functional conceptions.

I shall give diagrams in the endeavour to employ every possible means of exhibiting the tenour and tendency of my mode of view, and in the belief that a presentation in black and white of the schemata and of the symbols which have served as a standard of meditation, will help to expose the drift of reasoning—necessarily in large measure psychological, and therefore liable to extravagance of abstraction.

A principle that has greatly contributed to my thought-guidance is the notion that every neural process, even the lowest, is on the type of the psychological process of inference. I have gradually associated this notion with doctrines proceeding from empirical psychologists, more especially from Wundt, Bain and Fechner;¹ viz., from Fechner the relation between stimulus and sensation, from Bain the law of relativity, from Wundt the doctrine of neural inference. Without undertaking an adequate (and therefore lengthy) exposition of the relation knitting together these several topics, I must now content myself with a bare indication of the kind of relation which appears to me to exist, enough to justify the schema of inference which has served as one of my guiding notions. I will do so as briefly as possible in the following paragraphs:—

It appears to me to be very essential to recognise the applicability of the law of relativity in the most elementary sensation as well as in the higher provinces of judgment. In Fechner's theory a sensation is a comparison between two stimuli; the proper symbol indicative of its objective composition, is therefore a ratio $\frac{a}{b}$, and not any absolute quantity a or b .

I have gradually come to adopt the logical expressions: *major premise*, *minor premise* and *conclusion*, in analysing the factors of the simplest sensation, as well as of the most com-

¹ I name only those sources from which I have consciously derived instruction. I notice that James states that Wundt and Helmholtz have more recently "recanted" (i.e., retreated from the neural inference position). As far as my reading of Wundt goes, this seems to me a very gratuitous and misleading supposition on the part of Professor James. ("Psychology," ii. note to p. 111.)

plicated mental judgment. This is not intended to imply that the syllogistic terms of logicians correspond to any physiological factors in a neural process, but is rather the consequence of early associations, and of subsequent experience of the great convenience of this mode of formulation as regards the detection and localisation of fallacy in particular cases. And I think that the use of these terms is perfectly compatible with an arrangement of factors in the form of an equation, which although in substance identical with the three syllogistic propositions, appears to me more closely physiological than these as regards the representation of the neural process of inference.

But at this juncture it is of particular importance to exercise all possible care to avoid confusion between sensation—the subjective phenomenon, and sensificatory change of state, its imaginary objective substratum. And it is to guard against this fallacy that in symbolising the phenomena I find it useful to reserve Greek letters for the subjective and Roman letters for the objective state.

The simplest present sensation σ is not the concomitant of any isolate present state of organ S , but the resultant of a comparison based upon state now and state before now, *i.e.*, of a ratio $\frac{s}{s'}$.

The organic state now is the sum of many previous alterations of state; many sensations σ the concomitants of many previous ratios $\frac{s}{s'}$, may be conceived as summed up in the state of sensibility Σ .

This state of sensibility Σ has as its organic basis a material state, which (having regard to the total Σ by many elementary σ , each concomitant with a ratio $\frac{s}{s'}$) is properly represented by the ideal symbol $\frac{s}{s'}$.

Thus Σ the specific sensibility, the subjective resultant of past experience, is the concomitant of $\frac{s}{s'}$, the imaginary organic sum of the antecedent series of organic ratios $\frac{s}{s'}$.

To this resultant state (subjectively Σ objectively $\frac{s}{s'}$) I apply the term “personal ratio,” meaning to connote in this expression that the personal state now is composed of compared objective phenomena and not of *absolute* objective

But this description has only represented the process starting from given major and minor premises; it does not represent the mode of formation of a major premise, nor exhibit what in the process of sensation or judgment must be conceived as the order of procedure. A very few words will however suffice.

The starting point of the growth of sensibility is the ultimate fact "sensation," or the minor premise; sensibility or the personal ratio is the resultant of many past sensations or minor premises, *i.e.*, centripetal induction or synthetic generalisation of particular experiences. The starting stimulus in each act of sensation or of judgment is a sensificatory minor premise, and the consummation of a sensation or of a judgment is a compounding of premises in consciousness, attended with what can only be indicated as an intuition of parity or disparity between the psychological factors on the two sides of an equation, *i.e.*, major, minor on one side, conclusion upon the other.

I do not wish to press this symbolic mode of representation too far, yet I feel entitled to urge the picture as a useful one in bringing home to us the essential similarity of neural process concomitant with the whole range of subjective phenomena from the simplest sensation to the most complex judgment. It is in my opinion most congruent with the derivation of voluntary from reflex action, and with a physiological conception of the mechanism of ratiocination. It would be absurd to say that this or any other mode of symbolisation "explains" judgment, attention, sensation, but it is not too much to say that it clearly exhibits the possible factors of neural inference; each act of observation, each determination of conduct, is the resultant of two factors (1) the major premise or central state, more or less ad-tended to: (2) a particular minor premise or group of sensificatory stimuli, also more or less ad-tended to (within the remaining sensificatory field, less or more ad-tended to.) And in any theory of ratiocination, we must recognise that the primitive mental process is one of centripetal induction, its immediate consequent one of centrifugal deduction, and all higher mental processes on this reflex type compounded

of centripetal induction of minor premises with centrifugal deduction from a major premise.¹

Considering the centripetal aspect of neural processes we may with advantage appropriate to the three-level scheme the three terms *impression*, *sensation*, *perception*—using “impression” as the lowest-level term, “sensation” as the middle-level term, “perception” as the highest-level term; and taking impression to denote an effect that does not reach consciousness, sensation (= *empfindung*) to denote a felt impression, perception (= *wahrnehmung*) to denote a sensation in its felt circumstances, *i.e.*, in relation with the group of associated sensations forming the mental picture of the moment (= *vorstellung*). The relative significance of these more or less abstract and figurative terms may be assisted by comparison with a more concrete case. Taking cerebral cortex as the analogue of retina: the mental picture or representation or cerebral field of attention is analogous with the retinal field of vision; the perception is the analogue of the distinct object in the centre of the field, sensations the analogues of surrounding objects within the field viewed indistinctly; impressions the analogues of surrounding objects in the field that are not seen, although they can be seen. This indeed is rather more than an analogy, there is actually an association between physiological field of vision and psychological field of attention; retinal field is really retino-cerebral field; the great psychological importance of visual investigation is attributable to this association as well as to the fact that the visual field is the more complex and more susceptible of experimental analysis than any other of our sense fields.

¹ Readers of Spencer will probably recognise the close similarity of this mode of representation with that contained in the “Principles of Psychology,” vol. ii. I have not consciously borrowed them from that source, yet had I not there found the equation between two ratios urged as a more natural mode of representing ratiocination than the syllogistic form of logicians, I do not think I should have ventured to promulgate such a symbolism, although strongly persuaded of its greater fidelity to physiological phenomena. “Every ratiocinative act is the indirect establishment of a definite relation between two things, by the process of establishing a definite relation between two definite relations.”—Spencer’s “Psychology,” vol. ii., p. 16.

I have long been in the habit of making use of the following table and schemata, and have found them of service in guiding my thoughts. I have refrained from publishing them, believing the considerations embodied in them to be admitted as truisms, and feeling no call to add another verbal skeleton to the literature of the subject. But having used them and finding that they have led me to certain peculiarities in the mode of view, I find it necessary to expose them to criticism, as forming an introductory basis of what I believe to be a (in some degree) rational reading of cerebral psycho-physiology.

GENERAL ATTRIBUTES OF THE CEREBRO-SPINAL CENTRE.¹

I.—REFLECTION.—Every central element is an organ of return of action. It is excitable-exciting.

II.—RESISTANCE.—Each central element has a specific capacity for centrifugal tension resultant from centripetal impressions.

III.—RADIATION.—Centripetal impulses to a central element radiate to connected elements.

Inferior impressions influence superior centres; superior expressions influence inferior centres, in accordance with degree of resistance and strength of impulse.

IV.—RETENTION.—After each period of activity and restoration there remains an altered constitution of central elements, in which resistance to homogeneous activity is diminished, to heterogeneous activity increased.

These four R's embrace all the phenomena that came under my consideration, but I subsequently supplemented them by the following article, which is in reality an amplified statement of Article IV. The fundamental phenomena of exhaustion and replenishment (although constantly effective conditions of nervous processes) did not appear to me to belong to the present order of considerations, and are therefore not formally acknowledged here.

V.—SUMMATION (AND INHIBITION).—Two or more impulses summing in centrifugal tension or expression

¹ Each of the propositions enunciated on this page is considered to be preceded by the words "It is conceivable that . . ."

may be individually "friendly" or "unfriendly" to one another.

Homogeneous impulses or groups of impulses received simultaneously or successively by the same central elements produce a sum of effect greater than the effect of a single impulse or group of impulses—*i.e.*, are "friendly."

Heterogeneous impulses to the same central elements produce a sum of effect smaller than, or different from, the effect of a single impulse—*i.e.*, are "unfriendly."

Any two or more impulses or groups of impulses, whether homogeneous or heterogeneous, successive or simultaneous, upon different central elements, may be friendly or unfriendly; they are more commonly unfriendly.

The terms "impression" and "expression" are used above in a very general sense to denote centripetal impulses from the periphery towards a centre, and centrifugal impulses from a centre towards the periphery.

The impression to a superior centre is constituted by the impressioned resistance of an inferior centre.

Given three centres ranking I., II., III., and indicating impression, expression, and alterations of resistance or tension by appropriate letters and numerals. An impression I_3 enters III., produces an alteration R_3 and liberates E_3 . Altered R_3 constitutes impression I_2 , I_2 enters II., produces an alteration R_2 and liberates E_2 . Altered R_2 constitutes impression I_1 . I_1 enters I. alters R_1 and liberates E_1 . A second identical impression I_3 will meet with less resistance in III., it will produce a smaller change R_3 and conceivably a larger liberation E_3 ; a smaller change R_3 will constitute a smaller impression I_2 , therefore a smaller change R_2 , therefore a smaller impression I_1 , &c.

As the consequence of repeated identical impressions we thus have :—diminished resistance, the diminution being greatest in III., less in II., least in I.; diminished impression, the diminution of I_2 being greater than that of I_1 ; increased expression, the increase being greatest of E_3 , less of E_2 , least of E_1 , or otherwise: the minimum effective impression diminishes with repetition; the diminution is greatest in the case of I_3 , less in that of I_2 , least in that of I_1 , and we have as the

final result of repeated activity, greater diminution of R_3 than of R_1 , and a scale of most diminished minimum I_3 to least diminished minimum I_1 (with constant expression) of most increased E_3 to least increased E_1 (with constant I_3); which I take as signifying how, *With repeated exercise the reflex function specific to each centre, ranges in degree from most perfected in III. to least perfected in I.*



FIG. 1.

It involves these three assumptions:—resistance diminishes with repetition; impression ascending from lower to higher centre varies in the same sense as the resistance overcome in the lower centre; expression descending from higher to lower centre, or from lowest centre to periphery, varies in an opposite sense to the resistance overcome in the expressing centre.

The terminology used may prove a stumbling block to the word-picture I am attempting to compose. Impression and Expression are used in a peculiar and most general sense to denote impulse *to* and impulse *from* a centre. The diagram will, doubtless, make that sense plain. It contains this ambiguity. In the case of centre III. for instance, we have, properly speaking, two lines of expression *from* that centre,

one E_3 towards the periphery, the other I_2 towards the centre above. And to the same centre III. we have *two* lines of impression, one I_3 from the periphery, the other E_2 from the centre above. Thus I_2 is expression with reference to III., impression with reference to II.; E_2 is expression with reference to II., impression with reference to III. A similar ambiguity attaches to I_1 and E_1 . But it was necessary to make choice of symbols, and this has been done in accordance with direction to or from the highest centre.

I have employed the physical figurative term *resistance* after some hesitation, but no distinctly preferable term offered itself. Perhaps the words tension or elasticity come closer to the idea intended to be conveyed. We may imagine a centre as an elastic reservoir of tension, in which an ingoing impulse may be absorbed with increase of tension which may or may not express itself in an outgoing impulse. But to this physical term we must attach chemical connotations, considering that the impulse elaborates greater chemical instability or actual discharge. Thus "resistance" is equivalent to chemical "stability," diminution of resistance is diminution of chemical stability. But what now do we mean when we say that an impulse in passing through a cell "meets with less resistance," or produces a smaller alteration of tension, and how can we picture this in chemical terms? Such modes of expression obviously give a most imperfect picture of what actually takes place. An impulse does not properly speaking *pass through* a cell, it evokes elaboration within the cell, and in this view resistance is chemical inertia; but diminished resistance is not diminished chemical inertia, it is diminished chemical change or diminished chemical friction. I have said enough to indicate the imperfection of idea which is apt to be connoted by either a physical or a chemical figure of speech. Yet I have perforce made use of such a figure, viz., "resistance," choosing it as the most familiar, but guarding myself from the appearance of pushing the physical analogy suggested in the word.

The effects of an impulse upon a cell (indicated by the word alteration of resistance, and in the diagram by the letters R_1 R_2 R_3) I picture to myself somewhat as follows—

taking for illustration the centre III. The impulse I_3 influences cellular energy; it increases¹ potential energy, it may render potential energy actual; it tends to overcome or actually overcomes inertia, and in the latter case it effects friction. The actual effects of this cellular elaboration are the impulses E_3 and I_2 . In saying that a second impression I_3 meets with less resistance, I imagine that the cell matter is more unstable; this would seem to imply that the impulse would more easily disturb the whole mass of the cell, producing a more extensive change and thereby greater E_3 and I_2 . But I do not admit this. I imagine the greater instability of the cell to be *molecular*, so that the disturbance by a second impulse can propagate itself with less diffusion, overcoming less chemical inertia, and producing less friction, than the disturbance produced by a first impulse. The analogy with electrical resistance may make this point clear, but it is only an analogy. An electrical current entering by one point of a wet sponge of lower resistance would propagate itself with less diffusion, overcoming less chemical inertia, and producing less friction than in the case of a wet sponge of higher resistance.

Thus it is that I am led to imagine that the disturbance of a nerve-cell by a second impulse I_3 gives rise to a *greater* impulse E_3 and to a *smaller* impulse I_2 , than did a first impulse I_3 .

First impressions are the most intense in consciousness. Skilled labour is carried on with weak guiding impressions and weak consciousness.

It is more difficult to frame a diagrammatic representation of mechanism to fit our idea of synthesis, association, summation, inhibition. That which I am about to give is planned on a three-graded dichotomous type, and connotes what by many physiologists will be considered a false notion, viz., the representation of *different* peripheral parts in the *same* highest centre cell. Again, it assumes what anatomically we have no right to assume, viz., three levels of representative cells; all that we positively know as regards an anatomical

¹ For the sake of simplicity I leave out of view the anti-motor or inhibitory impulse.

justification of the schema, is that the total number of fibres between cortical and spinal cells is smaller than the total number of fibres between spinal cells and periphery.¹ Nevertheless, I have adhered to a three-level scheme as being most in accord with current psychological doctrine. And the representation of different parts in the same highest cell is in my view a material form of the psychological doctrine of the "unity of consciousness."

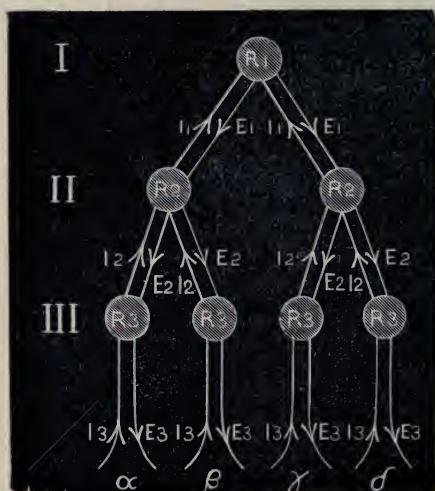


FIG. 2.

We cannot indeed make any sort of analysis of the centripetal process from impression to perception without imagining some sort of grading of nerve-cells; and the real anatomy of a hierarchic scheme is from this point of view quite subordinate to the ideal conception. The three terms adverted to above, viz., impression, sensation, perception, are as convenient and distinctive as any, and there is indeed no absolute necessity for adopting the further term apperception, which to some minds seems to possess very

¹ Sherrington's recent observation of "geminal" fibres in the spinal cord is perhaps another anatomical sign of an increasing number of fibres from above downwards above the spinal relay.

misleading connotations. The phenomena of hypnosis—more especially as presented to us in Bernheim's analysis—imperatively require us to adopt some such schematisation; *e.g.*, a hysteric hemianæsthetic, who asserts truthfully that she (or perhaps he) cannot feel, see, smell, taste or hear any impressions made on half her (or his) body, can be proved to have sensations of touch, sight, &c. He believes he cannot feel, and, therefore, does not feel, yet, all fraud excluded, it may be *proved* by appropriate devices that he does feel these sensations, but thinks that he does not do so, and, therefore, does not perceive them. Words are inadequate shortly to describe such a condition, yet it is an indubitable reality, and intelligible enough when regarded in terms of imaginary imprints on two different cerebral levels I and II. In the hysterical hemianæsthetic the imprint on II is effected, but the consequent imprint on I is travestied or untranslated owing to some functional disorder or incapacity of I, yet the physiological sequence from III to II and from II to I proceeds, and can be proved to proceed, if by any suitable device the false belief be circumvented—*e.g.*, such a person "blind" of one eye will see a real object doubled by a prism held in front of the sound eye; which proves that his "blind" eye is not "blind," that he has sensation by that eye when he does not think he is using it, but no sensation when he does think he is using it. Such a person may be and often is an impostor, but even a genuine sufferer would be set down as a malingerer in nine cases out of ten. Yet such a case might be perfectly genuine, the patient being at the same time "truly blind" of one eye, yet "truly not blind" of that eye, according to the sense in which we read these words "truly" and "blind." And the same may hold for any other sense; a genuine hysteric might be unable to hear or smell or move, because of her own conviction of impotence, yet able to hear or smell or move as soon as that conviction had been overcome. All these facts are best realised by a reference to two levels of cortical action II *sensory*, and I *perceptive*.

Although I shall refrain from entering upon a full and detailed consideration of the localisation doctrine, I can hardly

escape making some allusion to the obvious bearing of this upon the physiological doctrine of cortical representation, at the two opposite poles of which we have the "positive" contention of Munk, who experimentally verifies the projection doctrine of Meynert, and the "negative" contention of Goltz, who experimentally verifies the indifference doctrine of Flourens.¹ The antithesis thus shortly stated is at any rate as regards Goltz somewhat too absolute. Goltz indeed does not uphold absolute indifference, but rather an indeterminate and rudimentary predominance on a large scale and in the rough, of different function in different regions. His observations on dogs are irresistibly convincing; the observations of Munk upon dogs in proof of the projection of retinal upon cortical areas, require stronger confirmation than they have yet received before they can be admitted as real; Schäfer operating on monkeys confirms the principle in a very guarded manner, and describes the projection very differently from the view of it given by Munk on the same animal. In the presence of such mutually destructive data, we are at liberty—pending further developments—to attach weight to purely psychological considerations. In accordance with the doctrine of the unity of consciousness, we are called upon to believe that our perception of things seen, things heard, things felt, &c., may take place in connection with the modes of motion of a single organ.² Pictorial and verbal expression of this theory or belief is given by Wundt in his doctrine of a frontal organ of apperception; the views of Hughlings-Jackson that the frontal cortex is possibly the highest level centre, and of

¹ Many other names might of course be quoted; Ferrier, Schäfer, Horsley and Beevor, Charcot, Déjerine, and most clinical observers are "localisers" more or less strict. Brown-Séquard, Schiff, Dupuy, are "indifferentists" with more or less reserve. But there are many shades and degrees of opinion between the two extremes. Schäfer, for instance, is in some respects (G. Fornicatus) a localiser, but in others (hearing) an indifferentist; Exner is a "diffuse localiser;" even Goltz may be characterised a "diffuse localiser" inasmuch as he makes reservations to the complete indifferentism of Flourens; and the position held by Brown-Séquard and by Dupuy is very similar.

² "We see that a perception can have in a nerve-centre no definite localisation, but only a diffuse localisation. No one excited fibre or cell produces consciousness of an external object; the consciousness of such external object implies excitement of a plexus of fibres and cells" (Spencer's "Principles of Psychology," second edition, 1870, p. 562).

Ferrier,¹ that it is connected with the intellectual faculties, are in perfect agreement with this well-known Wundtian schema. Considering the state of probability constituted by physiological experiments and by psychological considerations, I find sufficient justification for adopting a neural schema symbolising a superposition of heterogeneous impressions and a common origin of heterogeneous expressions $\alpha \beta \gamma \delta$, in and from a single highest level cell I.² Considering, however, the probabilities as regards the cortical mantle formed of many such connected cells I., and the subordinate masses of cells II. and III. I adopt a view intermediate between the positive pole "localisation" and the negative

¹ Accurately speaking, this view is unreservedly adopted by Ferrier in the first edition only of his "Functions of the Brain," although it still remains in his second edition. But quotations will best serve to show the subordinate importance and indeterminate character of the anatomical localisation as compared with the ideal conception. Ferrier's present view is substantially identical with that of Munk, although Ferrier very justly in another place (p. 429) criticises and rejects Munk's nominal antithesis, "psychical" and "cortical." But, as has been remarked, Munk uses the terms in a naked denotative sense, meaning no more than might have been indicated by the terms α and β .

"The centres of inhibition being thus the essential factor of attention, constitute the organic basis of all the higher intellectual faculties. And in proportion to their development we should expect a corresponding intellectual power.

"In proportion to the development of the faculty of attention are the intellectual and reflective powers manifested. This is in accordance with the anatomical development of the frontal lobes of the brain, and we have various experimental and pathological data for localising in these the centres of inhibition the physiological substrata of this psychological faculty" (Ferrier, first edition, p. 287.)

"The hypothesis of Hughlings-Jackson and others, that 'in addition to the sensory and motor substrata which have been demonstrated and defined by physiological and clinical research, there are other and higher motor as well as sensory centres, in which all the motor and sensory functions are again represented, and form the substrata of the higher mental operations . . . receives no confirmation from the facts of experiment, nor does it appear to me at all necessary to explain the facts of normal or abnormal mentation. We have in the sensory and motor centres of the cortex the substrata of the respective forms of sensory perception and ideation, and of the individual acts of volition, simple and compound, as well as of the feelings associated with their activity. It seems more reasonable to suppose that there may be higher and lower degrees of complexity or evolution in the same centres than to assume the separate existence of more highly evolved centres, for which no evidence is obtained by the results of experimental research'" (Ferrier, second edition, p. 460).

Wundt rejects the view that our supreme perceptions of different sensations are separately consummated in separate centres, and characterises it as a revival of the phrenological faculty scheme. (*Phil. St.*, vi., p. 1.)

² It is, perhaps, hardly necessary to say that the expression "a single cell I." made use of on account of the diagram, must be taken to mean "a single mass of cells I."

pole "indifference." I do not think that I can express that position either more accurately or more shortly than I have already done elsewhere.

"The 'nerve-centre' of mammalia and of man is a collection of nerve-centres occupying the cerebro-spinal axis, with more or less diverse special offices under their control—communicating each with the other upon occasions, yet separately active upon other occasions—having functions which are localised at certain parts, yet not strictly confined to these parts—playing upon and influencing each other in all directions, yet in some directions rather than in others, and maintaining some kind of precedence and rank, so that while all may influence all, yet some are usually guided and controlled by others—variously organised through past excitations, yet still variously organisable by excitations to come.

"The state of doctrine [*i.e.*, of localisation], may be made plain by an analogy. The various activities making up the business of the brain do not all take place all over its surface, as in a country without towns or villages, where all kinds of industry go on in every hut or tent; nor are the different activities absolutely restricted to certain spots, as if in walled towns. The brain cortex is not comparable with either of these extreme cases; its territory must be recognised as possessing towns with special industries, but towns with straggling and overlapping suburbs, and industries which are indeed predominant, each in a given centre, but not exclusive of all other industries in that centre, nor excluded from other centres in which other industries predominate."¹

¹ My reading of Mosso's psychophysical observations (*Du Bois-Reymond's Archiv.*, 1890, p. 129), to the effect that prolonged intellectual effort subtracts from voluntary muscular workability, is in accordance with this intermediate view. If there are concentrations of different functions within an imperfectly differentiated organ, we should expect high-level exhaustion of any particular kind of activity to be greatest as regards that kind, but to exist in a minor degree as regards all other kinds of high-level activity. This, of course, applies only to the *central* component of fatigue, and not to the peripheral component attributable to the circulation of disintegration products. We may picture the functional depression as being of the entire cortex, but deepest at particular situations of concentrated activity. The converse change, viz., diminished capacity for intellectual effort after prolonged muscular exertion, is familiar to most brain-workers. On the other hand, most people have experienced that although one kind of mental activity fatigues the brain with respect to a different kind, yet such collateral fatigue is apt to be less marked than the principal fatigue, and so much so that a change of activity may feel restful.

And now, comparing the degree to which the functional and structural organisation has reached at different levels, I think we are justified in stating the following antithesis :

At the lowest level the representation is most definite and fixed, the canalisation of conductors is most closed, the differentiation of the central organ is most complete. At the highest level the representation is least definite and most fluctuating, the canalisation of conductors is most open, the differentiation of the central organ is least complete.

Contrasting the two extremes of cerebro-spinal action, we have in the purest reflex action an immediate, "fatal," unchosen response to peripheral stimulation ; the nervous path is deeply canalised, alternative reactions are few, the particular reaction to a particular stimulus is easily predicted. In the purest voluntary action we have a delayed, "free," chosen response to a given sensation. The nervous path is not preformed ; alternative reactions are numerous ; the particular reaction to a particular sensation cannot be predicted.

It is another and a further and an essential question whether the greater diversity of function and division of labour is of such degree in the human brain, that particular parts of the cortex have become the special organs of particular parts and functions of the body. And towards the answer to this question of degree, the deduction from experiments upon animals is of little value ; brain-surgery and clinico-pathological investigations of pure and uncomplicated cases of Jacksonian epilepsy and cortical monoplegia are the necessary and alone trustworthy premises of inference.

The localised effects of localised excitation of the cerebral cortex of dogs and of monkeys do indeed give fairly concordant results, which amply suffice to establish the principle that excitation of different parts of cortex and of subjacent corona gives different effects. But the converse test by localised extirpation has altogether failed to come up to the expectations that were first formed ; in the hands of Goltz extirpations have been made of larger and larger areas of the dog's cortex, of an entire hemisphere, and finally of both hemispheres, without producing permanent paralysis, while

in Munk's hands local extirpation of different areas on the same kind of animal (not to speak of monkeys) gave different local and restricted loss of sensation and of motion.

As is well recognised, the contradiction does not merely illustrate that different observers looking for different effects *see* different effects, but is in part owing to whether immediate and temporary defects or permanent deficit (Ausfallserscheinungen) were described as paralytic effects. Although *a priori*, a temporary paralytic effect is a possible result of a cortical ablation, we have no right to assume *a posteriori* that the defect is due to lost central matter; it may equally well be an inhibitory effect due to irritated central matter, as has been urged by Brown-Séquard and by Goltz.

And it is one of the fundamental principles of the Nancy school that the symptoms of an organic lesion include not only such as may be termed direct and organic, the immediate result of destroyed tissue, but also a varying and variable swarm of satellite symptoms, the secondary and functional effects of the primary. The organic blot is, so to speak, surrounded by a functional halo; and it is one of our greatest sources of doubt and difficulty that these fringe effects may, to use the language of Brown-Séquard, be effects of dynamogeny or of inhibition, and that we may even with extreme care be totally unable to distinguish and assign to their separate cause (1) the organic paralytic effects, and (2) the functional paralytic effects, the *ausfallserscheinung* from the *hemmungerscheinung*, total loss from functional suppression. It would therefore be most unsafe to reject Goltz's contention that a true localisation of function requires as its proof a permanent and not merely a temporary defect. And yet a doubt remains, even the lapse of time yields no sharp line of distinction; when shall we say "this is finally lost, this is at present suppressed," after Bernheim has taught us to recognise in human pathology that widespread and *chronic* functional modifications surround the organic lesion and its direct physical effects. Every physiologist recognises the physiological importance of the distinction urged by Goltz; some have also recog-

nised the meaning of Brown-Séquard's language, but for the present it must, I fear, be admitted that very few of us sufficiently recognise how that fundamental conception underlies the curative treatment of the Nancy school. The medical (and human) aspect in this department is the department of the pure physician, the physiological (and human) aspects concern the pure physiologist.

The principle of functional diffusion from an organic focus, foreshadowed in the clinical study of Jacksonian epilepsy, clearly evidenced in the comparatively simple but scanty data of the laboratory, comes to its full maturity in the hardly less simple but far more numerous and more minutely readable data insulated for us in the Nancy doctrine of so-called hypnotism. As "true localisers" we should expect to find a limb completely paralysed after its cortical centre had been destroyed, *e.g.*, in a dog. But this is not the case; the limb can be moved, and at first sight its movements seem perfect; it is certainly not completely paralysed. On closer examination some defect of movement may indeed be detected, some clumsiness of the limb indicating that its movements are inaccurately co-ordinated, and these imperfect movements will probably be found to be such as are normally most highly specialised and *not* such as are most profoundly automatic. But even this amount of defect is not apt to be permanent—the apparent awkwardness soon wears off, movements seem to be relearned. The clinical history of a dog or of a monkey having suffered a removal of some portion of the Rolandic area altogether negatives a strict localisation of function, and at most suggests its local concentration. We may imagine a company of cells more particularly concerned in particular manœuvres of particular parts, and we see these manœuvres rendered clumsy by disturbance of the company; we are obliged to imagine that even if these cells are wholly destroyed, the lost control may become more and more perfectly assumed by other cells educated from the periphery, as the clumsied movements are repractised, corrected, and learned. We thus picture the cortical organ in a semi-fluid state of differentiation, still variable by new instruction, rather than

as a petrified and invariable collection of specialist organs tied down to particular functions and exclusively performing these functions.

Such diversity of duty gradually superimposed upon uniformity of capacity, seems to me a sufficient physiological ground upon which to reconcile the assured results of cortical excitation, the hardly less assured absence of permanent paralysis after cortical extirpation in dogs, and the psychological doctrine of the unity of consciousness in man. The view is (I think) substantially that offered by Wundt in his doctrine of apperception, and whether we go on to hold with Wundt that the seat of supreme or maximum consciousness consists in a frontal organ of apperception, or with Munk that it is the central area of each particular *Fühlsphäre*, it seems to me that we must admit the cortex to be *one* organ, *qua* the ideas and concepts composed by impressions confluent through different afferent channels. Wundt's view, to my mind, gives clear diagrammatic expression to this necessity, Munk's view leads us into the admission of departmental consciousness; the first postulates one supreme consciousness, the second postulates many supreme kinds of consciousness. Although we may (and must) admit a physiological division of function and of organ up to a certain point, I think that in the present state of knowledge we need not (and therefore must not) admit any psychological subdivision of the supreme organ of consciousness.¹ To take a very simple case, we must admit that the notion of a bell is given to us in *one* auditory-tactile-visual organ, even though there be a discrete centralisation of auditory, tactile, and visual impressions. Nor does it seem to me to be of essential importance whether we admit that supreme "composite" consciousness is the attribute of a part of the cortex, or of the whole cortex acting as one psychological organ of confluence although composed of differing physiological departments. What does seem to me to be of essential importance is that a physiological division of function, ascer-

¹ The phenomena of "audition colorée" (viz., the association of certain colours with certain sounds, the latter "suggesting" the former) naturally come to mind in this connection.

tained by experiment, should not lead us to admit a plurality of supreme consciousness.¹

Guided by these considerations we may, I think, safely advance a step further in the *a priori* projection of thought:

The higher the brain the greater the mass of unformed formable material. But the further the differentiation shall proceed of formable material, the further it must already have proceeded of formed material. The higher brain must include subordinate highly specialised and economically-working mechanism-guiding parts. Otherwise it would not be free to be developed by new impressions and new combinations of impressions. And yet the further the brain has developed within the span of life of its possessor, the greater the differentiation of function and of organ out of the mass of formable material. We should expect greater concentration and fixity of function in the older brain, we find evidence of that greater concentration (with correspondingly dwarfed signs of dissimilar capacities) in the conduct of every matured and specialised man; experimenting on the cortex cerebri we should *a priori* anticipate clearer and sharper results from an old dog or an old monkey than from a young dog or a young monkey.² Thus it may be truly said, with reference to the functional attributes of the cerebral cortex that the higher the brain the less the fixity, the older the brain the greater the fixity.³

¹ Wundt has expressed this with great clearness in his recent criticism of Munk ("Philosophische Studien," vol. vi., 1890).

² Küssmaul makes the following apposite remark:—"One thing may be safely stated, however, and that is that the *age* of the patient is an important factor for the compensation of the injury. Children have been known to learn to speak after astonishingly extensive destruction of the left region of speech, and even of the entire cortex of the left hemisphere, while in old persons remarkably small foci of disease in the cerebral substance sometimes cause permanent aphasia." (Ziemssen's "Cyclopaedia," vol. xiv., p.803.)

³ The following quotation from Darwin ("Descent of Man," i. p. 38.)—"Little is known about the functions of the brain, but we can perceive that as the intellectual powers become highly developed, the various parts of the brain must be connected by the most intricate channels of inter-communication; and as a consequence each separate part would perhaps tend to become less well fitted to answer in a definite and uniform, that is, instinctive manner, to particular sensations or associations"—given by Dupuy as an opinion in support of the doctrine—"greater freedom of communication and of function in the higher brain," appears to me misleading in so far as the order of development is represented as being from the more special towards the more general, from heterogeneous towards homogeneous. The main drift of development exhibits itself as being towards specialisation, from the undifferen-

The schema given above (fig. 2) has, for several years past, furnished me with a concrete mechanical image to which I could refer the psychological phenomena of association.

If impressions of the two kinds α and β occurred simultaneously, and have been compounded in II., the repetition of α will be specifically liable to revive that compound effect which subjectively is the memory of α and β .

Similarly in the case of any other two or more kinds α , β , γ , δ , if their simultaneous or successive association in the past has been sufficiently frequent to leave a fused effect, the repetition of a single member α or β or γ or δ may revive that effect, *i.e.*, subjectively the memory of previously associated impressions.¹

The same schema (fig. 2) has also served to materialise my conception of the influence of attention upon particular incoming or outgoing impulses. I imagine an effort of focalised attention to consist in a state of increased tension along any single tract, α or β or γ or δ , together with a state of diminished tension along the remaining tracts $\beta\gamma\delta$ or $\alpha\gamma\delta$ or $\alpha\beta\delta$ or $\alpha\beta\gamma$. In which conception I tacitly assume the senso-motor character of the series of

tiated towards the differentiated; still it must be admitted as conceivable that secondary connections between more or less differentiated centres may become established, making *against* rather than *for* their functional severance; but the main and primary movement is disconnection, reconnection is secondary and subsidiary.

Herbert Spencer in the 1870 edition of his "Principles of Psychology" (and also in the 1855 edition), gives the clearest possible *a priori* account of the localisation question. Alluding to the phrenological scheme of localisation, he says: "Whoever calmly considers the question cannot long resist the conviction that different parts of the cerebrum must, *in some way or other*, subserve different kinds of mental action. Localisation of function is the law of all organisation whatever; and it would be marvellous were there here an exception."

"Either there is some arrangement, some organisation in the cerebrum, or there is none. If there is no organisation the cerebrum is a chaotic mass of fibres, incapable of performing any orderly action. If there is some organisation it must consist in that same "physiological division of labour" in which all organisation consists; and there is no division of labour, physiological or other, but what involves the concentration of special kinds of activity in special places."

"Among fundamental objections to this view, *i.e.*, the phrenologists," the first to be set down is that they are unwarranted in assuming *precise* demarcations of the faculties." ("Principles of Psychology," 2nd ed., vol. i., p. 573-4.)

¹ The Greek letters are not here used to denote subjective as contrasted with objective states.

cells I., II., III., and their susceptibility of undergoing augmentation or diminution of tension by impulses, *i.e.*, of undergoing alterations of excitability.¹ It is with this schema in mind that I am led to say, in somewhat figurative language, that "sensificatory stimuli affect us best when we meet them half-way by some action of our own, by an attitude of attention or by actual movements." In using such language I imagine a particular state of motion in I. causing in II. or in III. a state of increased excitability with regard to impressions homogeneous with previous impressions, of which the particular state of motion in I. is the material legacy.

§ 5—THE SENSE OF EFFORT.

I have on a previous occasion insisted upon the close connection between the family of sensations, individually denoted as sensations of movement, sensations of muscular contraction, muscular sense, sense of effort, and their after-effect, the sensation of fatigue. From the physiological point of departure the phenomena of effort open to objective study are neuro-muscular phenomena; but from the psychological standpoint the phenomena of effort extend over the far wider area of psychical experience, and are mainly, if not exclusively, approached by subjective study. I have said mainly, because, although in many cases the psychological presentation has been exclusively introspective and interpretative, yet in others it has included certain objective results of deliberate experiment—to give an instance—the psychical experience denominated "effort of attention" has been introspectively analysed, but what may be termed the phenomenal fringe of the subject, has been experimentally studied by psychophysical methods.

It was objected to my presentation of the matter that the general term effort was misused. Although I had not

¹ Everyone will naturally call to mind in this connection thought-reading tricks, and actual alterations of nutrition, brought about by the excessively focalised attention, which is the essential feature of the hypnotic state; the observations of Tarchanoff (*Pflüger's Archiv.* vol. xlv., p. 46) of local cutaneous electro-motive changes caused by attending to particular parts of the periphery, are also a physiological case in point.

specifically in view these forms of effort, I was alive to this use of the term; but I did not think it suitable to attempt to include them in the argument and I expressly limited that argument to the case of muscular effort. But so far from admitting that these more psychological forms of effort are to be dissociated from the most physiological form of effort, viz., muscular effort, I am disposed to insist upon the essential similarity of mechanism which runs through the whole range of effort experiences—sense of muscular effort, efforts of attention, of memory, of imagination, of will, of restraint. I can, however, do little more than offer an argument and state an opinion, appealing by the way to some few “experiments” (if experiments they may be called) which seem to indicate that an “effort of attention” is entitled to be spoken of as “nascent motor impulse.”

We may attend to something we are *feeling*, or to something we are *doing*, *i.e.*, we may apparently have a *sensory* or a *motor* or a mixed form of attention, this, however, is no valid distinction, for in attending to what we are *doing* it is evident that we are attending to the thing being done and felt as well as to the thing about to be done. So far then the phenomenon of attention is consequent upon sensificatory antecedents.

But it is not possible to have any great concentration of attention without some alteration of motivity—movement or arrest of movement—greater muscular tension or smaller muscular tension—and the so-called thought-readers illustrate this fact clearly enough. In attending to what we are feeling we always do something, *i.e.*, use certain muscles. The phenomenon of attention is an antecedent of motor consequences. A desire (*i.e.*, an imagined effect, *i.e.*, a remembered similar effect) is the antecedent and the consequent of movement. The psychological—I had almost said dialectical—dilemma, as to whether attention belongs entirely to motor neural processes or whether it is merely a form of sensation, does not seem to me a very serious one in terms of psychical phenomena.

Attention is “increased sensomotility,” at the same time

consequence of sensificatory antecedents, and antecedent of motor consequences. I use this word to denote that *one* property of a higher nerve-centre which has been improperly chopped into two properties, sensibility and motility of sensory and of motor centres. It is the homologue of the most general term excitability and of the most special term suggestibility. "Excitability," a name for the property of certain centres, motion with sensation. "Suggestibility," a name on the same pattern, from suggestion to conduct.

Let us review some cases to test the applicability of our definition—attention increased sensomotility. A chef d'orchestre picking out a weak instrument, a schoolmaster keeping an eye on a particular boy, a meditating monk or a howling dervish, a student in presence of a subject to be mastered, in short anybody doing anything, has his attention focalised upon his task in hand; we say that he concentrates his attention by voluntary effort; but if we imply that he originates the effort of will, we go beyond our tether.¹

It is most in accord with the principle "voluntary action

¹ I do not clearly understand Bastian's distinction ("On the neural processes underlying attention and volition") between the neural processes of attention and of volition, nor his conception of voluntary attention as "a compound or fusion of the two states volition and attention." The *two* names seem to me to denote *one* state or process or "alteration of sensomotility;" we have always diffuse sensomotility (states of attention and of inattention) and the plus state of sensomotility or "ad-tentive" state may explode in one volitional act or confine the explosion of other volitional acts; attention is nascent volition, volition is expressed attention; or otherwise, thought is nascent action. I cannot call to mind any example of purely sensory attention without motor concomitants; the indivisible double aspect of the process imposes itself upon me with such force that I am obliged to think of it under the compound word alteration of sensomotility. Not implying, however, that the process is to be regarded as compounded of sensory and motor factors, but to indicate that it exhibits subjective with objective symptoms—alterations of feeling with alterations of attitude—although itself a single and indivisible phenomenon. To the objection that we may have altered attention without altered movement, I would reply that such an apparent instance of pure sensory attention is a fallacy, and that an unseen motor consequence can always be made manifest as a visible motor consequence by intensification of attention without alteration of kind of attention.

To another possible objection that attention may have for its consequence not motion but arrest of motion, it is enough to reply that arrest of motion is as much a motor phenomenon as motion itself. If I attend to an object at the periphery of the field of vision, the cerebral process includes the production of an intensified fixation of the point of regard. If by an effort of imagination I reverse the position or relief of a transparent vessel, the cerebral process includes the production of an alteration in the diameter of

a disguised reflex" to refer attention, the psychological phenomenon, to increased sensomotivity, the physiological phenomenon. We can often enough in given cases discern the antecedent stimuli that may have elicited states of attention, but when we attempt to get behind the phenomena at the true cause of the state, we recognise that we are in presence of an ultimate property of cerebral living matter. We do not know.

We have, however, in the conditions and consequences of variations of attention, a field of exploration extensive enough to satisfy the most industrious aspirations to positive knowledge. Hypnotic phenomena, education, mental health and mental diet, in a word the influence of man upon man, are in the main determined through varying attention, varying sensomotility, varying influences by varying influences. And as a right theory is often the motive influence of right practice, so it becomes of great importance to acquire a right view of the physiological determinism of the phenomena of attention.

Much of our most valuable knowledge is analogical, one of the most valuable analogies with reference to attention, is afforded by retinal phenomena; the analogy is more than an analogy, for the retinal phenomena that we have to utilise are in reality retino-cerebral, so that a study of retinal

the pupil. Every antecedent has its consequences, every consequence has its antecedents, any phenomenon is a transverse section in the stream of change. Every centripetal impression has its motor consequences, every movement has its centripetal antecedents, any central neural phenomenon is a transverse section in the stream of neural change. We may call the stream past and future, centripetal and centrifugal, but the phenomenon itself is both or neither. I conceive the physiological phenomena underlying "emotion" as being similar in composition to those underlying attention, *i.e.*, as sensomotile, and not purely sensory nor purely motor, nor a resultant of separable sensory and motor factors. We know subjectively that an emotion entails feeling as a resultant of centripetal impulses, objectively that it entails movement or arrest of movement, the resultant of centrifugal impulses. Emotions are feelings expressed by motions, and the motor element is even more obviously manifested in emotion than it is in attention. But of the process itself we cannot say in either case that it is either *only* sensory or *only* motor; objectively imagined an emotion is the result of an alteration of nerve-substance, which alteration is the consequence of centripetal impulses and the antecedent of centrifugal impulses. And it is no drawback to the second half of this view, that the expression of an emotion can be restrained, for in restrained emotions there is the centrifugal tension or tendency, and it may be, actual movements suppressive of a natural muscular expression.

focalisation and of retinal contrast is in large degree a study of cerebral focalisation and cerebral contrast, which are among the most important phenomena of attention. I am aiming at one particular group of considerations that can, I think, with advantage, be presented from a very general point of view in this connection, viz., contrast phenomena.

Stated in general terms:—the value of any given constant is estimated to be + or — according as its surroundings are — or +; magnitudes are over or underestimated amid smaller or larger surrounding magnitudes, colours are intensified or weakened according as they are observed on similar or upon complementary¹ backgrounds; and psychologically any sensation is intensified or weakened according as it occurs upon a similar or a dissimilar field of attention. This law of contrast, simultaneous as well as successive, applies to the two portions of the field of attention which we have referred to as “distinct” and “indistinct”; and here again we may, with profit, represent by a ratio $\frac{\alpha}{\beta}$ the sensation resultant from its two component sensations α and β , α being the distinct area and β the indistinct area of attention. This resultant sensation will obviously vary with greater or smaller concentration of attention, according as the distinct component α forms a larger or smaller proportion of the total α and β . And now the law of contrast is evidently applicable; just as two antagonistic colour sensations mutually intensify each other, so do any two antagonistic masses of sensation; in this case the antagonists are the more intense distinct and the less intense indistinct portions of the total sensory field; the first diminishes the second, the second diminishes the first; in the hypnotic state the first annuls the second, and attention is at its maximum of concentration. Fixed attention is in fact the essential element of hypnosis of all degrees and however produced. In the normal state a person may be more awake to a particular kind of stimuli, less awake to all others; he is “pre-occupied,” and his pre-occupation may be trifling or moderate or considerable. In the hypnotic state it is excessive, the hypnotised person is over-sensitive to a

¹ Or antagonistic (Hering).

certain focalised portion of his environment, under-sensitive, or it may be impervious towards the whole unfocalized field.

In my previous paper (*Sense of Effort*, BRAIN, 1891), I offered as additional to the exclusively cerebral or spinal reflex view of movement, the following groups of considerations.

The initiation of action—Hitzig's image of movement, Munk's Gefühlsvorstellung or Bewegungsvorstellung, Ferrier's motor idea or impulse, Bastian's revival of kinæsthesis—is demonstrably attended with a consumption of force-producing material.¹ With this change manifested as diminished workability (motor fatigue) we have a sensation popularly termed fatigue (sensory fatigue), and I have urged above that the association of the two aspects is such as to forbid us to dissociate them; "motor" fatigue and "sensory" fatigue are one and the same thing, to which for want of a better word we may apply the term senso-motor fatigue. Moreover I have argued in my previous paper that the incidence of "fatigue" (which is the *result* of movement) may be taken as a guide to the incidence of "effort" (which is the *concomitant* of movement), and have concluded that "effort" and "fatigue" depend upon *changes at the centre and at the periphery*.¹

¹ This argument was the main motive of my paper. I find that James, who ostensibly pronounces himself in favour of the entirely peripheral origin of effort-sensation, commits himself to the position I take, viz., the distinction between peripheral and central factors.

"Effort of attention is thus the essential phenomenon of will. This volitional effort, pure and simple, must be carefully distinguished from the muscular effort with which it is usually confounded. The latter consists of all those peripheral feelings to which a muscular exertion may give rise ("Psychology," vol. ii. p. 562.)

Dr. Delabarre ("Mind," July, 1892, p. 383) objects that the fault of my reasoning lies in its major premise, which contains three unwarranted assumptions: (1) That the objective signs of exhaustion are always indicative of a previous expenditure of energy in the same parts., (2) That a subjective sense of fatigue is indicative of a corresponding previous effort and sense of effort in the same parts. (3) That objective signs of exhaustion are indicative of a subjective sense of fatigue, and objective signs of effort of a subjective sense of effort, localised in the same parts.

I really cannot discern these three assumptions in my major premise, which was clearly stated and printed as follows: *The material changes subsequent to action, are co-extensive in the motor organ with the material*

I admitted the peripheral sensificatory factor as matter of common knowledge, but offered evidence against the view that it is the sole factor of the sensations of motion to the exclusion of the concomitants of central emission. I also admitted as matter of common knowledge

changes accompanying action. The three assumptions are introduced by Dr. Delabarre rather than implied by me, and his objections to them are not objections to the premise itself.

This is a sufficient answer to the criticism, but I wish to clear the issue rather than to retrench myself behind a logical defence, and will therefore reply as follows to Dr. Delabarre's three assumptions:

(1) That Mosso's experiments (alluded to on p. 368 of this paper) do not appear to me to be at variance with the general truism that effects and after effects must have the same loci of incidence. The truism applies to the area of diffusion as well as to the focus of maximum at which the expenditure of force-producing material and the production of fatigue-products is presumed to occur, and experiments do not allow us to hold that the fatigue-products thrown into circulation can ever produce an effect at all approaching in intensity the local effect at the place of origin of such fatigue-products. (2) Delabarre's objection that the second assumption "is negated by the fact that the feeling of fatigue located in the eyelid is not at all commensurate with the amount of work this organ has accomplished, and is sometimes excessively strong in the morning, after complete repose of the muscles involved," introduces terms of comparison from which I carefully refrained, indicating as my reason that the introspective comparison of sensations is particularly liable to fallacy in this connection. I had and have nothing to say about any correspondence of magnitude between fatigue and effort, being well aware of the fact that the apparent magnitude of feeling varies with associated and contrasting circumstances. To trace any correspondence between two subjective magnitudes it is necessary to secure identity of circumstances and conditions. (3) The third assumption that objective signs of exhaustion may be utilised as indicators to a subjective sense of fatigue, although not contained in my major premise, is however made in the inference from premises to conclusion. So far from ignoring it and letting it smuggle itself unperceived into the argument, I repeatedly pointed out this assumption as the weak link in my chain of proof (Sense of Effort, p. 191, 192, 433). It was, so far as I could see, the one logical breach that might be employed by an adverse critic to break the proof, and I was careful myself to indicate that breach; I claimed no more than the *highly probable* parallelism between

sense of effort kinetic effect	and	sense of fatigue loss of kinetic power
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and therefore the highly probable character of the conclusion at the end of the inference. But I urge in defence of this breach three considerations: (1) That the weak link in my chain is no sign of the strength of any rival chain. (2) That "to take the demonstrable signs of material changes of objective physiological fatigue as indicators to the undemonstrable material changes of subjective psychological fatigue, keeps us closer to phenomena than the hitherto universally adopted procedure of drawing conclusions from the suggestions afforded by simple introspection." (3) That, although probability is not proof, yet proof is no more than overwhelming probability, so that in the opinion of some the high probability of my conclusion may be held as amounting to proof.

Dr. Delabarre himself grants as much, and rather more, in some introductory sentences that may be compressed as follows: "Granting that objective

that the various components of the sensificatory factor arising from skin, joints, tendons, and possibly muscle itself, must be perceived centrally through afferent channels. I see no reason for adopting the theory of Lewes, that our knowledge of motor change can be derived from centripetal impulses along the efferent tract.

signs of exhaustion are indicative of a subjective sense of fatigue, and granting that the exercise of central energy is indicative of a sense of effort localised where the effort takes place, the central sense of motor innervation is proven." And although it may be psychologically allowable to say that "we know without elaborate experiment that the central organ exerts energy," I, for my part, prefer to this *a priori* and indefinite premise, observed physiological data, *i.e.*, objective and measurable signs of central expenditure.

Professor G. E. Müller (*Zeitsch. f. Psychologie, u.s.w.*, 1892, p. 122), also adversely criticises my paper on the "Sense of Effort." The following sentence sums up his criticism:—"Verfasser schliesst auf die Entstehungsweise der normalen Empfindungen des Muskelsinnes ohne Berechtigung aus der Entstehungsweise der Ermüdungsempfindungen. Auf letztere schliesst er ohne Berechtigung aus der Entstehungsweise der objectiven Ermüdungserscheinungen, denen er ohne stichhaltige Beweisgründe einen vorwiegend zentralen Ursprung zuschreibt." That is to say, he rejects my data and my reasoning. As regards data, time and space do not allow me at present to review them in detail, but I shall take an early opportunity of doing so with reference to Prof. Müller's searching and therefore valuable criticism. I am well aware of their imperfection, and hope that other observers may assist in their experimental purification and prosecution. As regards reasoning I wish Prof. Müller had not gone outside the inference as formulated by me on p. 190. I urged a conditional or probable conclusion, and he represents me as claiming to have produced an unconditional proof. I had guarded myself from this claim as clearly as I was able, *e.g.*, on pp. 190, 191, 192, 433. I admit that the sequence considered by Prof. Müller to be "ohne Berechtigung," does fairly and correctly represent the view I held and hold, but I cannot accept the omission of the qualifications that I recognized to be necessary. The main object of my paper was to urge the examination of this complicated matter with the neuro-physical change (objective) rather than the psychical perception (subjective), as the point of departure. Whereas psychologically we have to deal with four (or more) distinguishable kinds of phenomena produced in voluntary movement, physiologically we have to deal with one kind of phenomenon, *i.e.*, the material (motificatory and sensificatory) change, and upon a balance of evidence and of probability to form an opinion as to the organic distribution of that change. Starting from the concept of a single neuro-physical change with kinesis as its objective expression, æsthesis as its subjective concomitant, I examine the kinetic power at the centre and at the periphery as the accessible objective indicator to the neuro-physical change producing kinesis and æsthesis. I do not see reason to admit that kinesis and æsthesis are produced by separate neuro-physical changes, still less to admit that qualitatively distinct æstheses in the same neural segment by the same cause (*e.g.*, sense of movement and sense of fatigue) are produced by separate neuro-physical changes.

The hypothesis serving as my guide may be put thus:—

- I. Neuro-physical change occurs with voluntary motion, (1) in the cortex, (2) in the spinal cord, (3) in the end-plates, (4) in the muscle, (5) in the tendons, articulations and skin. 1 and 2 are central, 3, 4 and 5 are peripheral. [For reasons given in my previous paper (p. 181) I exclude nerve-fibres from consideration. Nor is it necessary in the present connection to consider a sixth component, *viz.*, spinal and cortical

I do not share this view because I see no evidence to support it. Indeed, the supposition has appeared to me to be so purely gratuitous that I did not even mention it in my previous paper.

changes consequent upon 3, 4 and 5; these secondary central changes are admitted by every one, and to include them in the present field of thought might possibly distract attention from the point at issue, viz., the existence of primary central changes; whether such "primary" changes are truly primary, *i.e.*, uncaused, or resultant from antecedent centripetal impulses is another question that has already been considered in this paper.]

- II. The sensation of motion arises from neuro-physical change in one, some, or all of these situations.
- III. Neuro-physical change in consequence of voluntary motion, having as its objective sign physiological or "objective" fatigue, occurs in 1, 2, 3, 4 and 5.
- IV. The sensation of fatigue arises from neuro-physical change in one, some, or all of these situations.

I find evidence (criticised and rejected by Müller) that objective fatigue is central and peripheral, I infer that the neuro-physical change, with motion and after motion, is central and peripheral. If the sensation of fatigue is the subjective concomitant of that neuro-physical change, then subjective fatigue has central and peripheral sensificatory factors. If the sensation of movement is the subjective concomitant of the neuro-physical change, then the sensation with movement has central and peripheral sensificatory factors.

As far as I can see, the acceptance or rejection of my reasoning from the phenomena of objective fatigue to the distribution of its neuro-physical basis, and from the latter to the distribution of the neuro-physical basis of objective movement, and of the sensations of fatigue and of movement, must in last resort depend upon a preference between the following conceivable alternatives. The objective movement and the subjective sensation of that movement depend upon the disintegration of *one* neuro-physical substance S. M. or of two distinct neuro-physical substances S and M. The two alternative con-

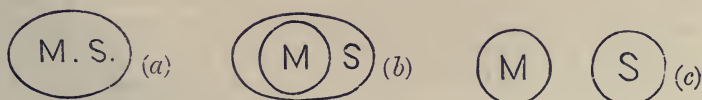


FIG. 3.

ceptions may be symbolised by the (a) and (c) in fig. 3. An intermediate alternative in which all neuro-physical matter is regarded as sensificatory, but only some such matter as also motor (some being only sensificatory) may be symbolised by (b) in fig. 3. I regard concept (a) as the most probable, concept (c) as the least probable. Concept (b) which represents that material other than inogenic may contribute to æsthesis, is in this connection nearer to alternative (a) than to alternative (b), if we recognise that every sensificatory change influences centrifugal tension. Subjective fatigue and the sensation of movement depend upon the disintegration of *one* neuro-physical substance or of *two* distinct neuro-physical substances. I regard the former as the more probable alternative. "*Neither more, nor more onerous, causes are to be assumed than are necessary to account for the phenomena.*"—(Hamilton.)

Dr. Bastian invokes Gotch and Horsley against it in the following terms:

“The lack of sensibility accompanying the action of motor centres has now been experimentally demonstrated by Gotch and Horsley.”

I do not know whether Dr. Bastian has fully appreciated the gaps and pitfalls between Gotch and Horsley's data, and the conclusion which he draws from them. But in so far as he is a disbeliever in a retropulsive wave along an efferent tract he is, in my opinion, justified. The Bell-Majendie experiments on nerve-roots prove this point, by demonstrating that it is not possible (in the absence of recurrent sensory fibres) to obtain a reflex effect by stimulation of the central end of an anterior root, or by stimulation of a mixed nerve after section of the posterior roots.

Gotch and Horsley,¹ although they have not gone so far as to claim to have “experimentally demonstrated the lack of sensibility accompanying the action of motor centres,” have indeed drawn a conclusion which does not seem to me to depend upon the data from which they draw them, but upon other and far more assured and familiar data. They consider that:

“As the electrical method affords the first opportunity of an experimental contribution to this subject, it is interesting to find how strongly its application bears out Bastian's position. In this chapter it appears probable that the kinetogenetic portion of the centre is the afferent [*i.e.*, that in which the potential energy is converted into kinetic] side of it, and the more especially when it is seen how readily the centre discharges into the afferent nerve channels (*i.e.*, actually ‘backwards’ as compared to the course of ordinary afferent impression).”

“The method has further enabled us to ascertain what connections and facilities for conduction the efferent, or so-called motor side of a nerve centre possesses, and instead of finding, as might have been expected from the ordinarily expressed beliefs on this subject, that we have to deal with a source of energy that was readily aroused, and freely connected with its neighbours, we found, to our surprise, that it afforded nothing of

¹ Croonian Lecture. *Phil. Trans. R.S.*, 1891. Summary of Cap. xi., p. 509).

the sort, and that its power of conducting impulses centripetally was practically *nil*.

"Curiously enough, this last point was foreseen also by James, who in his celebrated work on the 'Feeling of Effort,' 1880, while endorsing the views of Bastian, says that 'the electrodes of the physiologist,' if applied to the central end of the anterior root, would not arouse any 'sentient,' *i.e.*, afferent impulse in the cord. We are happy to find that our experimental results, unusual though they were, are, nevertheless, in close agreement with the deductions of the logical method of these distinguished writers."

I do not dispute either the electrical data, or the conclusion that impulses do not reach the brain from muscle by running up efferent tracts, but I do not think that the latter is borne upon the former; to my mind the facts indicate an altogether different kind of conclusion, and the conclusion is established from far more direct and simple data. As regards the second point, it is enough to remark that the absence of movement on stimulation of the central end of an anterior root—forming part of the ordinary class demonstration of Müller's experiment on the nerve roots of the frog, and of Majendie's classical demonstration of the same fact on mammalia—proves it conclusively. As regards the first point, while it is possible that the facts may be congruent with this established truth, it is equally possible that they may illustrate quite a different matter; it is well known that the negative variation in nerve is elicited in far greater amount by electrical stimuli of nerve than by either functional or mechanical stimulation;¹ doubt has been felt and expressed whether the "negative variation" and the nerve-impulse are co-extensive phenomena;² the large variations up and down the uninterrupted fibres of the posterior roots and columns, as compared with the small variations down the anterior roots and the absent variations up the cord

¹ DU BOIS-REYMOND. "Thierische Elektrizität," vol. ii., chap. 6, p. 507. Electrical excitation gave a deflection of 40°, whereas non-electrical excitation gave only 1° to 3°, and the powerful functional tetanisation produced by strychnia gave 1° to 3° or even 4° deflection.

² KUHN. "Zeitschr. f. Biol.," xxii., p. 339, 344 *et seq.*, in admitting "double conduction" in nerve, bases his conclusion upon the results of his gracilis experiment rather than upon the evidence by negative variation.

above the anterior cornual cells, suggest that they are dealing with an electrical phenomenon depending (at least in part) upon the physical continuity or discontinuity of fibres, rather than with any pure and simple electrical *measure* of the magnitude of functional disturbance in the nerve. That this last assumption (*i.e.*, measurement of functional impulse by its electrical indication, or properly speaking by galvanometric deviations) is improbable, is further indicated by some of the authors' own results. They obtained large effects down the afferent tract with centrifugal discharge, and diminished effects down the efferent root; in most cases this centrifugal discharge was by electrical excitation, but in a few cases it was functional discharge; these few cases are the real key to the validity or the reverse of Gotch and Horsley's contention that nerve-impulses pass freely down (as well as up) the afferent tract and root, diminished down the efferent root from the cord, and not at all up the efferent root to the cord. But even if the contention be admitted as valid, it is difficult to see what additional cogency as regards the kinæsthesia doctrine would be added to the far more cogent facts (1) as to the afferent root, the known failure of response by excitation of its central end, and (2) as to the posterior root, the known passage of centripetal impulses along it. The non-existence of an effective repulsive wave *up* the efferent tract is proved by (1). The existence of a repulsive wave *down* the afferent tract (as urged by Gotch and Horsley), interesting as it may be, would add no strength to our knowledge of the centripetal transmission of peripheral sensifactory stimuli.¹

It is difficult to understand how any logical thinker can be led to translate this maze of inference into "an experimental demonstration of the lack of sensibility accompany-

¹ If more proof were needed of the non-existence of a repulsive wave from muscle up motor nerve we might adduce the observations of Kühne, who altogether failed to find any evidence of its existence in experiments made *ad hoc* upon the frog's gracilis ("Zeitschr. f. Biologie," xxii. p. 333).

Du Bois-Reymond, discussing the evidence of double conduction afforded by the negative variation, remarks that the absence of reflex response to stimulation of the central and of a motor conductor is intelligible on the assumption that the central stations of motor nerves are inexcitable by the repulsive wave ("Thierische Elektrizität, ii., p. 574).

ing the action of motor centres." It will be very much to be regretted if this verbally clear but phenomenally obscure dictum should be uncritically quoted as a "datum experimentiæ;" I have therefore done my best to disentangle the argument and to make plain its possibilities and probabilities of fallacy.

This has been a side issue; so far from having led into it, I deliberately ignored it in my first paper after the most careful consideration; but as it has been raised in authoritative quarters (in a presidential address to the Neurological Society, and in a Croonian Lecture to the Royal Society), it had to be entered upon.¹

I have also advanced what I consider to be a very important extension of view with regard to the neuromuscular government of the body, viz., that in addition to

¹Bastian, until quite recently (BRAIN, 1887, p. 44) quoted Wundt and Bain as the exponents of the cerebral emission theory. I pointed out (BRAIN, 1891, p. 436) the injustice of attributing to Wundt an opinion expressly repudiated by him, and signified adhesion to Wundt's real opinion, as contrasted with Bain's. And Bastian now (BRAIN, 1892, p. 2) makes a similar distinction between Bain and Wundt. This is the passage upon which I had relied as regards Bain's position:—

"As the nerves supplied to the muscles are principally motor nerves, by which the muscular movements are stimulated from the brain and nerve-centres, our safest assumption is that the sensibility accompanying muscular movement coincides with the outgoing stream of nervous energy, and does not, as in the case of pure sensation, result from an influence passing inwards, by ingoing or sensitive nerves. It is known that filaments of this class are distributed to the muscular tissue, along with the motor filaments; and it is reasonable to suppose that by means of them the *organic* states of muscle affect the mind. It does not follow that the characteristic feeling of exerted force should arise by an inward transmission through the sensitive filaments; on the contrary, we are bound to presume that this is the concomitant of the outgoing current, by which the muscles are stimulated to act. No other hypothesis is adequate to represent the total opposition of nature between states of energy put forth, and states of passive stimulation."—Bain, "Senses and Intellect," 2nd ed., 1864, p. 92.

But I now learn from Prof. Bain that he also admits the participation of a peripheral factor.

So that, after all, it is permissible to bracket Bain and Wundt, not as maintaining the exclusively central source of the sense of effort, but as holding it to be of mixed origin; the two names need not be separated. I have hopes that it may eventually be allowable to add Bastian's name to these; for the present, however, this addition is not permissible, he still holds to the exclusively peripheral origin of the sensifactory changes with *muscular* effort, and distinguishes this form of effort as differing in kind from effort of attention, volition, &c., of which he presumably admits a *central* material substratum. Of course, by saying that Bastian holds to the exclusively peripheral origin, &c., I do not mean to imply that he ignores the truism that sensation is *consummated* centrally. But for the present he holds that the sensation of movement (kinaesthesia) is *initiated* solely at the periphery, whereas Wundt (and Bain) hold that it is initiated at the centre, and at the periphery.

(1) central initiation,¹ (2) reflex response, we must admit (3) direct response, in the perfect quantitative co-ordination of muscular movements.

This third item was very cursorily alluded to in my previous paper in a note to p. 244, and opportunity may therefore be taken to more adequately develop it.

The view that reflex spinal action plays a very large part in the government of muscular action is very generally accepted, and the extent to which spinal control is held to be effective has of late years been greatly augmented. Among published papers I may quote Exner's² and Chauveau's.³ I may add that in 1880, when the question occupied my thoughts very largely, I was strongly inclined to the view that reflex muscular action responsive to the extension of muscle or tendon or ligament must be a chief factor in the co-ordination of muscular movements. I still admit this view, but not to the extent that I might have done, nor as unreservedly as is done by Exner, Chauveau and many others, owing (1) to the fact which I demonstrated in 1880,⁴ and (2) to the important principle long ago discovered by Heidenhain,⁵ but even now scarcely appreciated at its full value by neurologists or by physiologists.

(1) The positive item which withheld me from supposing that muscular co-ordination is altogether by reflex action from the cerebro-spinal axis, was the phenomenon of "tendon-reflex," which is demonstrably not a reflex from the cord, but a direct phenomenon of which a continuous reflex tonus is a necessary condition. The phenomenon being recognised as a direct response to an extensile excitation, we cannot fail to recognise the direct excitability of muscle itself by sudden tension, as well as its indirect excitability

¹ This word might be taken in an active or in a passive sense, to imply a spontaneous or a provoked initiation of nerve-impulses. It is here taken in the second sense in accordance with the physiological postulate: voluntary action = a species of reflex action. But see also the concluding paragraph of this paper.

² EXNER. Ueber Sensomobilität. *Pflüger's Archiv.*, xlviii., 1891, p. 592.

³ CHAUVEAU. On the Sensorimotor Nerve-circuit of Muscles. *BRAIN*, 1891, p. 145.

⁴ "On Muscular Spasms known as 'Tendon-Reflex.'" *BRAIN*, 1880, p. 179.

⁵ HEIDENHAIN. Mechanische Leistung, Wärmeentwicklung und Stoffumsatz bei der Muskelthätigkeit. *Leipsig*, 1864.

viâ a central controlling organ, as being contributory to a balance of power between muscles, variously stretched by each other's contraction, and as supplying a most rapid corrective of any sudden fault of balance.

And this direct responsibility being recognised, we cannot fail to see that all that is said in exposition of the reflex adjustments of position by reflex contraction responsive to tension, applies with additional force and even clearer "zweckmässigkeit" to direct adjustments of position by direct contraction responsive to tension.

The principle may be summed up in the short dictum—*ut tensio sic contractio*. As has been said the visible contraction, in response to a direct extensile stimulus, requires, as a necessary condition, that subsensible degree of reflex action, which, for want of a better name, is called reflex tonus, and if the reflex arc is cut at any point the direct contraction fails. But the dictum "*ut tensio sic contractio*" may be discovered to apply also in some degree to muscle isolated from the nervous system.

(2) The principle of muscular action contained in Heidenhain's often quoted but little known monograph, published nearly thirty years ago, may be summed up in this double proposition:—*ut tensio, sic labor, sic calor*.

The proof that muscular work increases with muscular tension is contained—I had almost written concealed—in every text-book of physiology. Up to a certain limit an excised frog's muscle does more work in contraction the greater the extending load, and within a somewhat narrower limit the actual contraction or shortening increases with the load. Thus we have illustrated the dictum "*ut tensio sic contractio*" in the accurate sense of the word *contractio*, *i.e.*, amount of shortening, as well as the dictum "*ut tensio sic labor*." It is important to recognise now that in our first use of the dictum "*ut tensio sic contractio*" we have not used the word in its accurate sense but have included in it connotations which properly belong to the word "*labor*." When we summed up the principle of direct response to extension in the words "*ut tensio sic contractio*," this last word implied amount of work as well as degree of shortening in

the muscular contraction, and the proposition "*ut tensio sic labor*," which as we have seen applies to excised muscle, might therefore have been substituted for it.

Heidenhain's observations show further that the increased work with increased load does not occur at the expense of the other form of actual energy manifested by contracting muscle, viz., heat, but that with increased load there is increased heat as well as increased work. *Ut tensio sic calor* as well as *ut tensio sic labor*.

These relations teach us that, other conditions being constant, but with variable load or tension or resistance to contraction, the amount of energy manifested by isolated muscle is variable. The strength of contraction (*labor*) varies with the mechanical task to be accomplished; more weight or more tension or more resistance to contraction elicits a larger production of muscular energy in the forms of work and of heat, the isolated muscle evolves energy in proportion to demand by tension.

This is the second feature to which I have referred to above as the "direct responsibility" of muscle, a property which in my opinion plays an important part in the accomplishment of exact movements appropriate to varying states of tension.¹

§ 6—APPERCEPTION.

We have already had occasion to refer to Wundt's theory of Apperception (p. 372). It is indeed impossible to make any progress in the analysis of cortical function from either a physiological or a psychological point of departure without

¹ The principle appears to me to be of such importance that we ought to be particularly careful not to admit it uncritically, but on the contrary to expressly recognise the limited extension of the heat-data that form its chief basis. Heidenhain's conclusion is exclusively based upon experiments with excised frog's muscle; attempts to verify the principle with mammalian muscle made in the Leipsig laboratory by Lukjanow (*du Bois-Reymond's Archiv.*, 1886, suppl. p. 117) yielded in this respect a perfectly negative result, the principal *ut tensio sic calor* could not be experimentally verified. On the other hand it is one of the principal conclusions of Chauveau's researches on human muscle (*Le travail musculaire*, Paris, 1891), but in this case a critic acquainted with the subject might easily find technical loopholes for objection. Another point of deficiency that can only be filled in by thermo-electric experiments of extreme delicacy is the failure of proof of any difference in amount of heat evolved by the extension of living *versus* dead muscle.

involving oneself in a consideration of that theory. The nature of the problems of which it is offered as an explanation, is such that the explanation itself requires much further explanation, and that it has been read or imagined as conveying a meaning diametrically opposed to the whole spirit and tendency of Wundt's teaching. No one has abjured faculty fetishism in psychology more plainly and explicitly than Wundt, yet we find him repeatedly pointed at as a faculty-monger and triumphantly refuted.

Of recent years this misrepresentation has been particularly noticeable in England, and judging from a review of Münsterberg's *Beiträge* by Dr. Titchener, the fashion seems to have been set in Germany.¹ Yet, making the most liberal allowances for the intrinsic difficulty of the subject, and for the natural preference by a hurried reader for second-hand allusions written in English to the original expositions written in German, the travesty remains an amazing one to anyone acquainted with Wundt's writings. To go back no further than the beginning of this year, we may refer to the last presidential address to the Neurological Society as an instance in point. Wundt's "so-called faculty of apperception" (p. 20) is there alluded to and refuted, but to the reader acquainted with Wundt's writings there is hardly any trace of Wundt in the so-called Wundtian position there presented and demolished. Indeed it is only too clear that this authoritative contradiction, which may not unreasonably be expected to influence opinion in this country, is based upon the case as presented by Wundt's adverse commentators rather than upon that contained in Wundt's own declarations, and the Wundtian position carried by Dr. Bastian seems to me to represent not what Wundt thinks, but (on p. 20) what Croom Robertson thinks Münsterberg thinks Wundt thinks, and (on p. 15) what Bain thinks Wundt thinks. The bald faculty puppet presented to us

¹ "Dr. Münsterberg's polemical aim is the demolition of a *soi-disant* 'theory' or 'metaphysic' of Apperception. Like pulpit atheism, this theory is a thing of straw, which the author sets up and knocks down with a wearisome monotony. The serious part of the matter is that he has fathered the 'metaphysic' upon Wundt." (*Mind*, 1891, p. 530.)

by Bain¹ and by Bastian as Wundt's child, was long ago torn to pieces by Wundt himself² (*Phys. Psych.* 1886, p. 354). This much is simple matter of historical fact to be verified or refuted by reference to Wundt's classical work.

Wundt's position appears to me to be in the main similar to that of Munk³ (α and β cortex function) (with the difference already alluded to in the footnote on p. 333) and of Jackson (high-level and middle-level cortex function) and to that which is presented in § 4 of this paper. Indeed my own views have been in large part formed by reflecting upon a hint originally derived from Wundt, *i.e.*, the notion of neural synthesis. But of this I have already spoken. I hasten to add that in giving this reading of Wundt's attitude *re* apperception, I do not pretend to make Wundt responsible. The misconceptions liable to arise in any transfer of abstract thought from mind to mind, have multiplied around these words—will, attention, thought, apperception—and the association of contradictory connotations round the same words in different minds, have almost destroyed their value as middle terms between mind and mind.⁴ Yet I feel obliged to offer my interpretation for whatever it may be worth; it

¹ Wundt. (*Phys. Psych.*, 3rd ed. 1887. Vol. ii., p. 389, note 7). "Den seltsamen Bericht, den Alex. Bain seinen englischen Lesern von dem sonstigen Inhalt meiner psychologischen und metaphysischen Ansichten gibt, kann ich hier mit Stillschweigen übergehen. Er beweist, dass dieser ausgezeichnete Gelehrte seine Kenntniss meiner Psychologie nicht dieser selbst, sondern dem Bericht irgend eines Lesers verdankt, der die Lectüre meines Buches mit seinen Reminiscenzen an irgend einen Cambridger Theologen des 17 Jahrhunderts verwechselt zu haben scheint."

² Wie hat sich doch die neuere Psychologie mit der Ueberwindung auch der letzten Spuren der obsoleten Vermögenstheorie gebrüstet! (Wundt, *Phil. Stud.*, vi., p. 17). The term "faculty," in consequence of its adoption by the phrenological inventors, has acquired connotations that have altogether unfitted it for employment except as a polemical missile. Otherwise it often presents itself as a most suitable term to denote particular psychological properties; "memory" as a psychological faculty of a special cerebral organ is a physiological non-sense; sight, smell, as physiological faculties or susceptibilities to particular forms of excitation (*i.e.*, in a passive sense with the connotations of the French word "faculté," as implying a potentiality, *e.g.*, *faculté d'agir*) would be in many cases a suitable and most convenient term, if only the word had not been debauched. But as it is, the word is only fit to be used as an abusive label for psychological vagaries. Perhaps it may become disinfected by time.

³ As regards historical order the two names should, of course, be transposed.

⁴ A. Marty remarks incidentally that "es bedarf natürlich einer ganzen Reihe von Verwechselungen und Aequivocationen um von hier aus [*i.e.*, Apperception = das Bemerken] die Apperception für identisch mit dem Willen zu erklären. (*Zsch. für Psychol. und Physiol. der Sinnesorgane*, 1892, p. 140).

is at any rate the resultant of a strenuous "effort of apperception" excited by Wundt's own writings.

The points of *opinion* aroused in me by the careful perusal of Wundt's exposition (that in the 3rd edition of his *Phys. Psychol.*, 1887, as well as that in the 2nd edition (French trans., 1886), are :

1st. That he altogether rejects the faculty theory of cerebral activity.

2nd. That "apperception" is no more than his label to denote high-level neural synthesis.

3rd. That Wundt's doctrine of apperception is in no opposition to the associationist doctrine of the English school (the two Mills, Bain, Spencer), but fully inclusive of that doctrine.

4th. That Wundt's frequent indentification of "Apperception" and "Will" illustrates the impossibility of dividing the supreme central process into sensory and motor. "Apperception" (the term with centripetal connotation) and "Will" (the term with centrifugal connotation), are thus distinct terms denoting one indivisible senso-motor process of "Thought."

But this is only my opinion, and I am open to correction in this somewhat obscure and dubious matter. All that I venture to assert positively is that Wundt has been misrepresented to English learning by the paraphrases of Bain and of Bastian, precisely as Kant was misrepresented to a former generation by the careless eloquence of Coleridge. The Kantian faculty puppet was fathered by Coleridge, not by Kant; the Wundtian faculty puppet now slipping into English literature, is equally illegitimate.

It is not possible to describe the highly abstract bearings of the Wundtian doctrine of "apperception" by any short paraphrase, or by quotations extracted from their context. It is not possible in this case to get anywhere near the author's meaning without careful study of his own writings. With the express reservation that short quotation is in this case insufficient, I will select some passages in which Wundt's meaning seems most to come to a point, although point apart from shaft is not a very convincing instrument.

The last two references taken from a more recent paper, are given to indicate what indeed may be found indicated at greater length in the *Physiologische Psychologie*, viz., the great extension given by Wundt to the term "Will," and his view that reflex action springs therefrom, and not *vice versa*—a view shared by many leading psychologists, among others by Delbœuf, and—taking the spontaneity doctrine in its broad sense—by Bain.

"Den Eintritt einer Vorstellung in das innere Blickfeld wollen wir die Perception, ihren Eintritt in den Blickpunkt die Apperception nennen."—Vol. II. p. 236.

"Weiterhin muss aber sogar die Apperception als der primitive Willensact angesehen werden, der bei den äusseren Willenshandlungen stets vorausgesetzt wird. Bedingung für die Ausführung einer Willensbewegung ist die Apperception der Vorstellung dieser Bewegung."—Vol. II. p. 243-4.

"Ein wesentlicher Theil der Schwierigkeiten, welche zu jener Annahme einer Entwicklung des Willens aus den Vorstellungen geführt haben, verschwindet sofort, wenn man die Apperception als die primitive Willensthätigkeit anerkennt. Von einer Zeit der Willenslatenz, in der sich erst die Vorstellungen, welche eine Beherrschung der äussern Bewegung möglich machten, im Bewusstsein ansammeln müssten, kann dann an und für sich nicht mehr die Rede sein. Die innere Willensthätigkeit ist von Anfang an mit dem Bewusstsein gegeben, da es ein Bewusstsein ohne Apperception nicht gibt, und die äussere Handlung erscheint als eine Bethätigung des Willens, welche von der inneren Handlung der Apperception nur in ihren Folgen, nicht aber in ihrer unmittelbaren psychologischen Beschaffenheit verschieden ist. Als Phänomen des Bewusstseins betrachtet besteht nämlich die äussere Willenshandlung in der Apperception einer Bewegungsvorstellung. Die wirklich erfolgende Bewegung und die daraus entspringende weitere Wirkung auf Bewusstsein und Apperception ist erst ein secundärer Erfolg, welcher nicht mehr ausschliesslich von unserm Willen abhängt: die Apperception der Bewegungsvorstellung oder der Willensentschluss kann erfolgen, ohne dass die Bewegung eintritt, sobald der Zusammenhang der physischen Werkzeuge, die bei der Bewegung zusammenwirken, in irgend einer Weise gestört ist."—Vol. II. p. 470.

"Da aber weiterhin die Contraction des Protoplasma sauf den späteren Entwicklungsstufen deutlich den Character eines psychophysischen Vorganges an sich trägt, d. h. einer physiologischen

Leistung, welche zugleich von psychischen Vorgängen begleitet ist, so verlangt der Grundsatz der Continuität aller Entwicklung, dass dieser Character auch schon jenem primitiven Spaltungs—und Contractions vorgang nicht fehle : er wird in diesem Sinne als ein einfacher, von Empfindung und Gefühl, eingeleiteter und begleiteter Willensact zu deuten sein " (*Phil. Stud.*, 1889, p. 339).

"Jede Uebung besteht in der Mechanisirung ursprünglich mit Bewusstsein geübter Willenshandlungen" (*Phil. Stud.*, 1889, p. 378).

And the senso-motor view seems to me very explicitly set forth in the page on which these sentences occur :—

"In jedem Gefühl ist eine Willensrichtung, in jedem Wollen eine Gefühlswirkung enthalten das Gefühl ist das erste Stadium eines jeden Willensactes. . . ." (*Phil. Stud.*, 1890, p. 376).

I cannot pretend to have fully mastered Wundt's meaning; his treatment of apperception as the determinant of Will, and in some places his apparent use of the two terms as inclusive of each other, and above all his presentation of will as the fundamental property of living matter, are depths of doctrine to which I cannot claim to have penetrated. My reading of Wundt's adoption of "Will" as the fundamental protoplasmic neurility, is that it gives expression to the fact that the functions of living things in their lowest, as in their highest manifestations, are not *finally* explained by reference to passive properties, that "excitability" and "association"—the last words of physiology and of psychology—are not the last words of philosophy. He appears to prefer to denote his foundation concept by the active term WILL rather than by the passive term EXCITABILITY. To me the choice seems fundamentally indifferent, and that the same dead end to positive knowledge has been reached whichever term we adopt. The truth that "it is the spirit that quickeneth" seems to me as clearly apparent beyond "*excitability*" as it is in "*will*," and it is merely a matter of verbal convenience to us as students of phenomenal determinism, whether we adopt "Excitability" as our foundation concept, and say that voluntary movement, objectively studied, is reflex movement; or whether we take "Will" as our foundation concept, and say that amoeboid

movement, subjectively idealised, is voluntary movement. The inspiration of all *living* matter is equally manifest to us, whether we look through man to protoplasm and see in amœboid movement the first signs of "will," or through protoplasm to man and see in conduct the highest expressions of "excitability."

ON THE DESTINATION OF THE ANTERO-LATERAL ASCENDING TRACT.

BY HOWARD H. TOOTH, M.D., F.R.C.P.

IN the present volume of *BRAIN* (p. 215) Dr. Mott contributes a very interesting paper on the upward course of the degeneration which follows lesion of the antero-lateral region of the white matter of the cord. Some of the material on which that paper was written was brought before the Physiological Society at King's College Laboratory on March 12th, 1892. At that meeting I stated that I had experimental and anatomical evidence of similar results to those of Dr. Mott, and that I was about to bring my results before the next meeting of the Neurological Society.¹ The experiment had been made in the Laboratory of the Examination Hall in December, 1890, and it is owing to the kindness of the Laboratories' Committee that I am enabled to make the following communication.

I had drawn attention to Loewenthal's experiments and observations on dogs in my Gulstonian Lectures in 1889 (pp. 62 & 64), and I had at that time also traced the degeneration of the antero-lateral ascending tract as high in the medulla as the points of emergence of the sixth and seventh cranial nerves (*op. cit.*, fig. 14), but had failed to trace it any higher. This was no doubt owing to the initial lesion (a semi-section) being too low down in the cord, the degenerated fibres being too sparse at the higher levels to be recognised by the ordinary Weigert-Pal staining method. Dr. Mott's experiment consisted of cutting the fibres of the tract by a specially-devised knife in the upper cervical region. He thereby caused a marked secondary degeneration, which was made all the more evident by the method of staining he employed.

¹ Held at University College Physiological Laboratory on March 17, 1892.

In the experiment about to be detailed the lesion included the direct cerebellar tract as well as the antero-lateral ascending tract, and in that respect must be held to be of less value than Dr. Mott's, but owing to the height at which the lesion was made (lower medulla) the degenerations are quite unmistakeable by the Weigert-Pal method of staining.¹ The appearances resemble almost exactly those in Loewenthal's drawings.

Though I am unable to trace the fibres with certainty to their ultimate destination in the cerebellar cortex, I consider that the diagram given by Dr. Mott on p. 219 represents the anatomical relations of the antero-lateral and direct cerebellar tracts correctly, as far as their course in the medulla, pons, and central white matter of the cerebellum is concerned.

On December 6th, 1890, the following operation was performed on a bonnet monkey. The medulla was exposed by trephining the occipital bone on the right side, the circle of the trephine hole touching the edge of the foramen magnum. The remaining sharp edges of bone, and also the lamina of the atlas, were removed by the bone forceps. The dura was then carefully slit up so as to expose the medulla and inferior vermis of the cerebellum. A small dorso-ventral incision in the lateral region on the right side was then made by means of a fine cataract knife. All this of course was done under strict antiseptic precautions and under complete anæsthesia.

The animal recovered so rapidly as to be practically quite well and lively the next morning.

Sensation was tested in all forms (except the clip test) and found to be perfect.

The animal was killed thirty-four days after the operation. After hardening in Müller's fluid the medulla and cerebellum were imbedded in celloidin, and the sections were stained by the Weigert-Pal method.

Microscopical Examination.—The general appearances are represented diagrammatically in the plate, which is a

¹ Dr. Mott and I have compared our results carefully, and have agreed that the degeneration in the two series of sections are practically identical.

reduction of the diagram shown at the meeting of the Neurological Society at University College, on March 17th, 1892. The degenerated areas are indicated by black patches.



FIG. 1.—Through the medulla above the level of the first cervical, and at about the middle of the lesion. The lesion is seen to be a very small one. It involves the direct cerebellar and antero-lateral ascending tracts. It has involved a very few of the fibres of the ascending root of the fifth (V.a.).

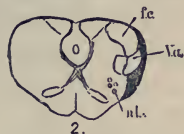


FIG. 2.—At the level of the lower olive. The initial lesion is no longer to be seen, the appearances being now due to true secondary degeneration. This consists of a strip extending dorsally from the funiculus cuneatus (f.c.), to ventrally the level of the nucleus lateralis (n.l.) It bounds externally the ascending fifth (V.a.), which is normal. Many normal external arcuate fibres may be seen streaming into the degenerated area to form the corpus restiforme higher up. Many degenerated fibres may be seen in the nucleus lateralis.

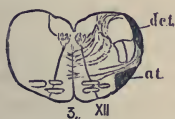


FIG. 3.—Made through the lower rootlets of the twelfth nerve (XII.). Central canal now opened out into the fourth ventricle. The strip of degeneration is now practically divided into two parts, a dorsal portion situated externally to the funiculus cuneatus, intensely degenerated, which is the direct cerebellar tract (d.c.t.), and a ventral, situated externally to the nucleus lateralis, which is the ascending antero-lateral tract (a.t.) These two tracts are divided by a neck of healthy arciform fibres belonging to the external series.

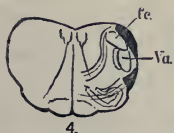


FIG. 4.—A little above the last. The degeneration of the direct cerebellar tract is becoming invaded by numbers of normal fibres derived from the external arciform group, the two together forming the corpus restiforme. The ascending antero-lateral tract consists of many degenerated fibres lying among the external arciform fibres dorsally and externally to the olive.



FIG. 5.—At the lowest part of the eighth nerve (VIII.), and the ninth nucleus and nerve (IX.)

The direct cerebellar tract degeneration is now a small patch in the dorsal third of the corpus restiforme, which is here well developed. The antero-lateral ascending degeneration is compact, and lies just dorsally to the olive.



FIG. 6.—At the upper extremity of the olive. Corpus restiforme now very large, with the small direct cerebellar degeneration in its dorsal third. Antero-lateral ascending tract in same position as before. (VIII. a.) ascending root of eighth nerve.

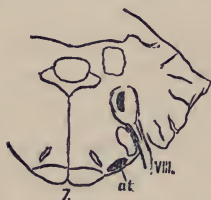


FIG. 7.—At the level of the seventh nucleus and superior olive. The corpus restiforme is quite buried in the mass of the middle cerebellar peduncles, and still shows its small patch of direct cerebellar degeneration. Unfortunately here and in the next section there is a considerable amount of decomposition evident in the central parts of the section; this is due to the slow penetration of the hardening reagent.¹ It requires very careful comparison between the two sides to be sure which is degenerated and which is decomposition.

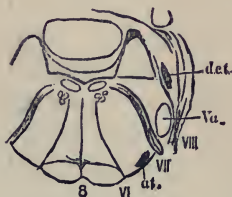


FIG. 8.—Intra-medullary course of sixth (VI.) and seventh (VII.) cranial nerves. The fibres of the corpus restiforme are at this level cut obliquely owing to their taking a direction dorsally to pass to the vermiform process of the cerebellum. The degeneration of the direct cerebellar tract can still be seen, but there is also some decomposition change in its surroundings. The brachium con-

¹ I have endeavoured to get over this by injecting the whole animal with Müller's fluid through the aorta, after washing the blood out by neutral saline solution immediate after death. The Müller's fluid, however, causes so intense a spasm of the small arterioles that the injection cannot reach the central parts. This difficulty is obviated by sending a fairly strong solution of atropine through the vessels, after washing out with saline solution, thus causing paralysis of the muscular coats of the small arterioles.

junctivum, or superior peduncle of the cerebellum, is now seen in somewhat oblique section with the fibres of the corpus restiforme curling round it. The two brachia conjunctiva form the side walls of the upper part of the fourth ventricle, stretching across the ventricle and forming its roof is the velum medullare anterius, dorsally to which is the lingula or upper extremity of the vermiform process. The velum medullare anterius consists of white fibres apparently arising from the brachia. In the angle formed by the brachium with the lingula is a small mass of degenerated fibres cut in transverse section. These are the bent-back fibres of the antero-lateral tract. So in this and the succeeding figures, except the last, we see the two limbs of the loop of the antero-lateral ascending tract—one ventral and the other dorsal.

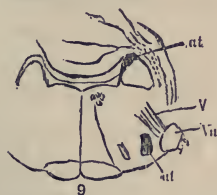


FIG. 9.—At the level of the upper part of the loop of the seventh nerve. The brachium conjunctivum is now well differentiated; it consists of a main body of transversely cut fibres, somewhat pyramidal in section, with a sort of cap of

obliquely-cut fibres applied to its apex and mesial aspect. The set of fibres on the apex is deeply degenerated (a.t) as stated in the last paragraph, the mesial set is continuous with the velum medullare anterius. Hooking round the brachium are the fibres of the restiforme body, which appear to go to the lingula, and many are degenerated, the direct cerebellar tract (shown by dotted lines). The ventral part of the antero-lateral tract (a.t) is beginning here to take an oblique course ventro-dorsally towards the trunk of the fifth nerve, which is here just sending off the fibres which are to make up the so-called ascending root.

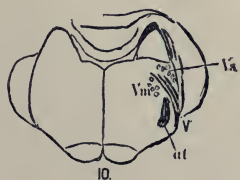


FIG. 10.—At the level of the fifth nerve (V.) The disposition of the dorsal degeneration of the antero-lateral tract remains the same, being at the apex of the pyramidal section of the brachium conjunctivum. The fibres of ventrally-disposed part (a.t) are now cut about longitudinally, and are

seen to be taking a direction as if to intersect the course of the fifth nerve. Vs. sensory and Vm. motor nuclei of fifth.



FIG. 11.—Just above the fifth. Dorsal part of antero-lateral tract, now disposed more on the outer aspect of the brachium than before, the ventral part having now hooked round the fifth is approaching the dorsal part, the intervening fibres being those of the lateral lemniscus (ll). Lemniscus or fillet (l). Brachium conjunctivum (bc.)



FIG. 12.—Section through upper pons. The two degenerations are now quite close together. Vd. descending root of fifth. Pl. posterior longitudinal fibres.

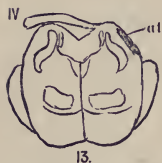


FIG. 13.—At the level part of the lowest point of exit of the fourth nerve (IV.) This shows the extremity of the loop formed by the antero-lateral ascending tract. The degeneration consists now of one strip applied externally to the fibres of the lateral lemniscus which lies between it and the brachium conjunctivum.

THE FUNCTION OF THE SYMPATHETIC GANGLIA.

A Reply to a Criticism by Dr. Friedric Vas.

BY W. HALE WHITE, M.D.,

Physician to Guy's Hospital.

I HAVE in various papers¹ urged that some of the sympathetic ganglia in adult man are functionless organs and are (like the appendix cœci or the coccyx) the remnants of structures having important functions in the lower mammals. With regard to the superior cervical ganglion this result was arrived at for the following reasons:—(1) The human adult superior cervical ganglia vary as much in size as do those of the largest and smallest of other mammals, but the size of the ganglion in other mammals than man varies directly as the size of the animal. It is well known that degenerate organs are particularly liable to vary considerably. (2) The superior cervical ganglia taken from forty-nine adult human beings were examined. Of these only one showed entirely normal nerve cells. In the others the cells were more or less shrunk, non-nucleated, granular and irregular in outline. They were also very strongly pigmented. The result of these changes was that often the cells were reduced to mere minute masses, usually of a bright yellow colour, lying quite free in the centre of the cell spaces, without processes, and as far as could be seen, without any connection with nerve fibres. But the superior cervical ganglia taken from five children showed that the cells were large, they filled their spaces, the nucleus and nucleolus were distinct, the protoplasm was non-granular, and there was no trace of pigmentation. The cells of the superior cervical ganglia taken from ten human foetuses were like

¹ *Journal of Physiology*, Vols. 8 and 10; *Guy's Hospital Reports*, Vol. 46; *Medico-Chirurg. Soc. Trans.* Vol. 68; *BRAIN*, Pt. 51.

those from the children. The facts appear to warrant the belief that the superior cervical ganglion although relatively prominent in the foetus atrophies with the growth of the individual. (3) I examined the superior cervical ganglia in sixty-eight mammals lower than man. Fifteen of these were monkeys and in four of them the cells were more or less like those just described as found in human adults, but in the remaining eleven monkeys and in the fifty-seven other mammals the nerve cells were well formed with a good nucleus and without any granular change, they filled their capsules and generally showed absolutely no pigmentation, although occasionally a little was present. Several of these mammals had been kept in the zoological gardens for many years and it was well known that they were very old, but yet their cells were quite different from the minute masses of granular pigment without nuclei which represented the nerve cells in the ganglia taken from old men. These results point also to the fact that in the mammals lower than man the ganglia are functionally active. (4) The examination of thirty human adult semilunar ganglia, of three from children, and of eighteen taken from mammals lower than man, for the same reasons as already urged for the superior cervical ganglion, appeared to show that in adult man the cells of the semilunar ganglia were inactive, but that they were functionally active in mammals lower than man. (5) The thoracic ganglia of twenty-four human adults were examined and it was found that in many of them the cells were large, distinct, neither granular nor atrophic, but in some these changes were slightly marked. It was concluded that in adult man the thoracic ganglia were only slightly degenerate. (6) Thirty-six gasserian ganglia taken from human adults were examined; in all the nerve cells were well formed with a distinct nucleus and nucleolus, and they filled their capsules. A few of the cells showed some granular pigment, but the cells themselves were not in any way atrophied. (7) Six lenticular ganglia from the orbit were examined. The cells all filled their capsules, were distinctly nucleated, but were neither granular nor pigmented. (8) Twelve spinal ganglia were examined. The cells never showed any of the extremely degenerate

appearance so common in the superior cervical ganglia, but often they were pigmented. (9) The cardiac ganglia were examined in ten patients; the nerve cells showed no shrinking, granular change, nor pigmentation. (10) It was shown that when the superior cervical and semilunar ganglia are invaded by disease no symptoms are produced except those which can be explained by the pressure on the nerve fibres passing through the ganglion, and that in the case of all diseases which have been ascribed to intrinsic lesions of the ganglia such a pathological explanation will not bear critical examination.

Thus it appears that the gasserian ganglion, the lenticular ganglion, and the cardiac ganglia are, in adult man, probably functionally active, and that most of the cells of the posterior spinal ganglia are also active. It will be noticed that the large number of 270 ganglia in all were investigated, and of each of these several sections were examined.

Dr. Friedrich Vas¹ has recently criticised the views I have put forward, and I propose to answer his objections.

Meyer, writing in 1876, suggested the view that some of the sympathetic ganglia in adult animals merely represented remains of structures which were of embryonal importance, giving as a reason that they could be excised from dogs without particularly affecting the animal from which they were taken. Vas urges rightly that we must not conclude therefore that they are functionless, for, as he says, the spleen can be excised and the animal will after a time be no worse for it. He might have also added that part of the brain and one kidney may be taken out without producing any permanent effect, but in all these cases it is quite certain that the excised organs have some function. But this just criticism by Vas of Meyer does not affect my contention at all, for an essential part of my view is that in dogs the sympathetic ganglia have some distinct function, but that in adult man this has disappeared. Meyer's argument is quite worthless either way, for while it certainly does not prove that the ganglia have no function, it certainly does not prove that they have any.

¹ *Allgemeine Wiener Medizinische Zeitung*, N. 45, 46 und 847, 191.

Vas states that I attribute great importance to pigmentation of the nerve cells of the ganglia, to their not filling their capsules, to their protoplasm being granular, and to the absence of the nucleus and nucleolus, as showing that they are a degenerate type of cell. This is not quite correct. I do not attach great importance to pigmentation as evidence of degeneration, I express myself very doubtfully on the subject. Thus (*Journal of Physiology*, vol. x., No. 5, p. 343) I say, "This may perhaps mean that we ought to regard pigmentation as evidence of degeneration." I never conclude because the cell is pigmented that therefore it is degenerate. It so happens that when from other reasons the cell is probably to be regarded as degenerate it is usually pigmented, and therefore when describing these cells I describe them as "granular pigmented atrophic" cells. I do not know the meaning of the pigmentation.

Vas goes on to say that I allow that sometimes nerve cells which show all these changes are nevertheless functionally active. I do not state this, and I never intended to convey such a meaning. Probably he refers to the fact that when the cells, which in any ganglion show these changes, are very few, I draw the conclusion that almost all the ganglion is functionally active, the only functionless parts of it being the few degenerate cells. Its activity is in fact just beginning to die out. This is the conclusion at which I arrived with regard to the thoracic ganglia and those on the posterior roots of the spinal nerves. In particular Vas mentions that I say that sections of the gasserian ganglion may contain one or two granular pigmented cells, and yet he objects that I do not conclude from this that the gasserian ganglion is in adult man an embryonal remain. Of course I do not. The very fact that the vast majority of its nerve cells were like ordinary healthy nerve cells goes to prove that nearly all the nerve cells are functionally active and I distinctly state those few which were altered were never atrophic. From these facts I think I am justified in my conclusion that the ganglion as a whole is functionally active.

He next states that there are two questions to be asked

in connection with my view. In the first place, if the sympathetic ganglia are embryonal remains (*embryonale Reste*) how comes it about that they continue to increase in volume after birth. My answer to this is that I never said that the ganglia were embryonal remains. I said that those ganglia which in adult man show degenerate cells (*e.g.*, the superior cervical) were "atrophied degenerate organs like the coccyx or appendix cœci." Embryonal remains are structures which in the life of the foetus were of great vital importance to it, such as the umbilical cord or the hypo-gastric arteries, and they will, directly after birth as their function then ceases, begin to gradually atrophy. But organs which are vestigial structures, representing in man organs which in the lower animals were important, and which are gradually dying out, will not necessarily begin to atrophy, because life changes from intra-uterine to extra-uterine. Such organs may continue to grow (although not to anything like the extent to which they do in the lower animals) for a long time in the life of the human being. For example, the coccyx, an undoubted degenerate organ, does not begin to ossify till birth, and the last centre of ossification does not appear till about the fifteenth year. Therefore, it does not by any means follow that because the superior cervical ganglion grows a little after birth that my view is incorrect.

The second question asked of me is, how is it possible that, if in the human adult the superior cervical ganglia have lost their function, those of the thorax in organic connection with them have maintained theirs? Surely there is no reason why they should not. We do not argue that the coccyx of man cannot be a degenerate organ because the lumbar vertebræ are well developed. For all we know, in creatures in whom the superior cervical ganglia are well developed, they subserve functions quite different from those of the thoracic ganglia—in fact, this is very likely, for the superior cervical ganglion is a collateral ganglion and the thoracic are lateral ganglia.

Vas next gives as a reason why the superior cervical ganglia cannot be looked upon as vestigial degenerate organs

that they show no fatty degeneration. But this proves nothing. Numbers of degenerate organs, which in man undoubtedly represent structures which in the lower animals were of great importance, show no fatty degeneration; for example, the appendix cœci and the coccyx.

He goes on to say that the fact that the nerve cells of what I regard as the degenerate ganglia are very much smaller than their capsules is to be explained by artificial shrinking, due to the method of preparation. I am sure this is not so, for three reasons: firstly, the lenticular and gasserian ganglia in which it was not present were prepared in precisely the same way as those ganglia, as the superior cervical, in which most of the cells showed it; secondly, many ganglia, such as the thoracic and posterior spinal ganglia, showed this shrinking in some cells but not in others; thirdly, the cells can often be seen shrunken in superior cervical ganglia, when they are examined fresh directly they are taken out of the body. Also after the examination of hundreds of sections I feel quite sure he is wrong when he says that the nucleus and nucleolus of the human superior cervical ganglion are intact and easy to make out. I have examined hundreds of sections from man in which this is not the case.

Vas next urges that my view cannot be correct because if it were the ganglia of the rabbit should be different from those of man, which he says is not the case. In the first place it should be noticed that he does not say what ganglia he examined in connection with this point, but judging from his paper he does not appear to have investigated any but the superior cervical, so I conclude that he refers to these. In the next place it would not follow that because the superior cervical ganglion of the rabbit resembled that of man that the ganglion did not represent an organ that in man had lost a function which was important in the lower animals, for it is quite possible that in both man and the rabbit the ganglion might be a degenerate vestigial remain. For example, both in man and in a Manx cat the tail is a rudimentary, but we do not therefore conclude that the rudimentary tail of man is not the degenerate representative of the tail of the lower animals. It so happens

that although my published papers contain an account of the examination of the superior cervical ganglion of sixty-eight mammals lower than man, not one of these animals is a rabbit; so since reading Vas's paper I have examined a number of sections of both superior cervical ganglia of a rabbit, and I can confidently say that very nearly all the cells are well formed nerve cells, showing none of the atrophic condition so common in the cells of the same ganglion taken from adult man. Some of the other mammals show rather larger superior cervical ganglion cells than the rabbit, but almost all those in this animal are entirely different from those quite commonly found in adult man. If I am correct and Vas is wrong we see that the rabbit forms no exception to the rule that the cells in this ganglion of animals lower than man appear to show that it is a functionally active organ, although in man its cells are inactive and degenerate.

Vas further says that on my view the cells of the sympathetic ganglia of the frog ought to be functionless, for they are strongly pigmented, but I have already pointed out that I do not regard mere pigmentation as evidence of degeneration.

He next goes on to argue that if the nerve cells of the superior cervical ganglion in man are atrophic degenerate structures, you ought, by cutting the nerves either side of the ganglion, to be able, in rabbits, to cause the ganglion cells to have a similar appearance to that which I describe as the atrophic condition in man. Accordingly he cut the nerve above and below the ganglion in a rabbit and killed the animal fourteen days after. But I maintain that it is obviously unfair to contrast the condition artificially produced in a rabbit in fourteen days with that which has probably been produced in man by forces operating for countless ages. What would be thought of the man who, because he could not in fourteen days, after cutting all the nerves going to the tail of a long-tailed monkey, make it shrink to the size of the coccyx of an adult man, concluded that the coccyx in man did not represent the tail of the monkey. We need not therefore, I think, stop to discuss Vas's description of the results he obtained except to remark

that it is strange that he found that only the cells in the periphery of the ganglion were degenerated after section of the nerves at the side of it.

Vas admits that in some cases he found the human superior cervical ganglia to be such as I have described, but he says that all the patients presenting this condition had arterio-sclerosis of their vessels, and he is inclined to draw the conclusion that there is a casual connection between the degeneration of the ganglion cells and the arterio-sclerosis. There can, I think, be no doubt that this view is wrong. A reference to the lists published in the *Journal of Physiology* (vol. viii., No. 2, and vol. x., No. 5) will show that the forty-nine adults whose superior cervical ganglia I examined died of the most various diseases, and that no relationship can be traced between the disease from which the patient suffered and the condition of the cells of the superior cervical ganglion. In connection with this subject I may mention that Da Costa and Longstreath (*American Journal of Medical Sciences*, July, 1890) record eight cases of chronic Bright's disease, associated with what they consider to be pathological changes in the semilunar ganglia. The ganglion cells were pigmented and granular, neither nucleus nor nucleolus could be seen, and they were often shrunken. These changes have no value in Bright's disease, for, as I have elsewhere recorded (*Guy's Hospital Reports*, vol. xlv., and *Medico-Chirurgical Society's Transactions*, vol. lxxviii.), I have often found these changes in these ganglia even when no chronic Bright's disease has been present.

Lastly, I would point out that Vas's researches cover too little ground. He appears to have examined no other ganglion than the superior cervical, and that only in rabbits and in man. He does not say how many specimens he looked at. No children, nor fetuses were investigated, and even in arterio-sclerosis, about which he says so much, the superior cervical ganglia were the only ones examined, and even they were looked at in three cases only.

It will be seen, therefore, that in spite of Vas's criticism I maintain my view, which is given at the beginning of this paper, as to the meaning of the sympathetic ganglia in adult man.

HYALINE DEGENERATION OF THE SPINAL CORD.

BY WILLIAM BULLOCH, M.B., C.M. ABERDEEN,

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FROM the scarcity of literature on this subject it would appear that hyaline degeneration in the spinal cord is not a common condition. Most of the recorded cases have occurred in conjunction with syringomyelia. Anna Baümler (*Deutsch. Archiv. für Klin. Med.*, 1887) refers to several cases; among them one by Hutin, in which the dorsal cord is described as being of a glassy appearance.

Langhans (*Virchow's Archiv.*, Bd. lxxxv.) quotes the case of a girl, aged 19, who had a syringomyelic cavity in her cord. The cavity had been formed by the burrowing of a homogeneous gelatinous mass between the elements of the spinal cord, so as to compress the latter and lead to their degeneration.

A full account of hyaline degeneration, accompanied by syringomyelia, is given by Berkeley (*BRAIN*, 1890, vol. xii., p. 460). In his case, besides the fissure the cord was found extensively infiltrated by some hyaline material, which was in greatest amount in the neighbourhood of the vessels. Several vessels were also found filled with material closely resembling the hyaline masses. In some parts where the hyaline had exuded in large amount, the axis cylinders had been compressed and had undergone degeneration. After a careful research into the reactions and site of the hyaline material, Berkeley comes to the conclusion that the "new product is a peculiar exudation from the blood vessels, which, after absorbing and destroying to a certain extent the surrounding tissues, underwent itself a further change and broke down with cavity formation, or in some places with rupture of the vessels and consequent hæmorrhage."

In the case about to be related the changes found in the cord strikingly resembled those found by Berkeley in his case, except that in the present the changes were much more intense. The case at first appeared to be a typical one of acute ascending paralysis, but there appeared later other symptoms which compelled a reconsideration of the diagnosis, and then it became apparent that the case was very complicated from a diagnostic point of view, and atypical.

A case of acute ascending paralysis has been recorded by Eisenlohr (*Virchow's Archiv.*, 1878, Bd. lxxiii. p. 73), in which, at the level of the third cervical nerve, a mass was found which was strongly stained with carmine, and it showed fine slits in it but no trace of cellular or other elements. The same material was also found in the pia mater, especially around vessels which ran into the substance of the cord, and also in the commissural region near the great vessels there. A similar appearance was also found in the upper lumbar region, and also in the medulla and pons, although to a less extent. Duchenne ("Selections from the Works of Duchenne," edited by G. V. Poore, M.D., New Sydenham Society, 1883, p. 129) relates a case of "*Diffuse general sub-acute spinal paralysis*," in which a *post-mortem* examination was conducted by MM. Charcot and Joffroy. They found the "cervical enlargement of the cord enveloped in the posterior two-thirds in a kind of fibrous muff the texture of which bore no small resemblance in aspect and consistence to the tissue of the cornea. . . . In the back horns the nuclei were embedded in an amorphous finely granular semi-transparent soft material. In other parts of the back horns the nuclei had disappeared and the amorphous substance there constituted centres more or less voluminous, clearly defined and circumscribed by a kind of non-resisting membranous zone."

Charcot ("Lectures on the Diseases of the Nervous System," second series, p. 166, New Sydenham Society, 1881), refers to a case of cervical spinal myelitis with pachymeningitis, in which "three long and narrow canals were found in the cervical enlargement parallel to the long axis of the cord. Two of the canals were found mostly filled

by an amorphous, transparent, finely granulated substance, which at certain points had become disintegrated and had left in its place more or less extensive lacunæ with more or less irregular borders. This same finely granular substance slightly condensed formed the parietes of the foci, and without any well-marked line of demarcation became continuous with the adjacent tissue, which itself for some distance presented the characters of granular degeneration."

The appearances described by Charcot correspond to a certain extent to those which were found in the case to be described, for in many places the hyaline substance with high magnifying powers appeared to be finely granular, and it is probable that the transparent amorphous material described in the recorded cases was of the same nature. In the majority of the cases thickening of the vessel walls and the transformation of the latter into some hyaline material was observed.

The case was remarkable, inasmuch as the nervous symptoms came on after the patient had been suffering from profound anæmia of the pernicious type. Spinal degenerations in the course of pernicious anæmia, especially in the posterior columns, appear to be not infrequent, but the symptoms are masked by the profound general weakness.

Lichtheim (*Berlin Klin. Wochenschrift*, 1889, Oct. 14) found abnormalities in the cord in every case he examined. In half of the cases apoplectic extravasations had occurred, giving rise to minute sclerotic foci, while in the other cases the posterior columns were found widely degenerated.

The clinical history of the case is briefly as follows:—

W. B., aged 51, a farm labourer, was admitted into the Royal Infirmary, Aberdeen, on July 10th, 1890, under the care of the late Professor Smith-Shand. He complained of shortness of breath, and swelling of the feet and face. He said he had enjoyed good health until two months previous to admission, and he was of steady habits, there being no history of alcoholism or syphilis. His trouble had come on gradually. On examination he was found to be fairly well nourished, but showed considerable œdema of both feet and ankles and also of the face. He was profoundly anæmic,

the face presenting a pale lemon yellow colour. The temperature was normal and also the urine, except that the colour of the latter was very pale.

There was a loud blowing systolic murmur, heard loudest at the apex and transmitted round to the left axilla, where it became inaudible. The second sound at the pulmonic cartilage was accentuated. There was no evidence of hypertrophy of the heart, and the pulse was weak.

Respiratory and alimentary systems appeared to be normal. Liver dulness normal. Splenic dulness was found to be increased.

Examination of the blood showed that there was great diminution in the number of the red blood corpuscles, which at the same time were much altered in size and shape. Microcytes and poikilocytes were found in abundance, and a few of the corpuscles known as Eichorst's corpuscles were also seen.

Flammiform hæmorrhages were present in both retinae, but no optic neuritis.

The nervous system appeared to be healthy except that in his intellectual functions the patient was slow and heavy of comprehension, and he had occasional delusions. The motor functions were undisturbed, although he was rather awkward and unsteady when he got up. At the time this was attributed to his great weakness, but in the light of the further development of his trouble it may have been the commencement of the spinal disease which manifested itself later on. From the state of the blood and the profound asthenia, coupled with the colour of his skin and the hæmorrhages in the retinae, the diagnosis of anæmia of the pernicious type was made, and he was put upon five-minim doses of liquor arsenicalis three times daily, and he was carefully dieted. Under this treatment he improved greatly, the blood becoming richer in corpuscles and the form of the latter being normal. The awkwardness of gait also disappeared, and with the return of strength the swelling of the feet also.

On the 12th of September, after a two months' stay in the Infirmary, he was sent to the convalescent hospital for a short stay previous to his return home. During his stay

here he became the subject of frequent hallucinations, believing that people were speaking to him and annoying him during his sleep. Notwithstanding these peculiarities he was perfectly manageable and caused no disturbance, and he regained his strength so much that he walked several times to the hospital to report himself.

In the third week of his stay at the convalescent hospital the superintendent observed that he exhibited great inco-ordination of gait, and that if he stood for a short time his legs gave way beneath him. The gait was when seen at this time ataxic, and he was unable to walk in a straight line. Romberg's symptom was also present. In addition to the ataxia, however, there was distinct paresis of the legs, and this became so great that on the 2nd of October, 1890, he was again removed to the hospital. On the following day the paresis had involved the whole of the lower extremities, and by next day the paresis had become paralysis. The limbs were flaccid, and superficial and deep reflexes were abolished. Bladder and rectum were unaffected. About the 5th of October the hands and arms became affected, and great inco-ordination for all movements was observed, rendering the patient absolutely unfit to feed himself or do almost anything with his hands. On the 7th paresis was noticed, and by the 9th of October both arms were completely paralysed. His mental condition was one of apathy and listlessness, and his memory began to fail so that he called people by their wrong names although he could recognise them perfectly.

His condition, which was one of complete motor paralysis of upper and lower limbs, remained practically unchanged for some weeks. The tactile sensibility was diminished. The sense of locality, position of the limbs, the muscular sense and the thermal sense were normal.

He sometimes passed fæces and urine involuntarily, but this appeared to be due to his general failure in intellect rather than paralysis.

His temperature, which had remained normal, used sometimes to rise as high as 104° F., and then come slowly down during the next two or three days.

His appetite became capricious, and he suffered greatly

from thirst. He had several severe attacks of diarrhœa, which lasted for three or four days at a time. On two or three occasions his urine required to be drawn off by the catheter. No pain was complained of during the entire course of the illness, the patient always describing himself as feeling better.

Towards the end sensation appeared to be affected, but the patient was so stupid that rational answers could not be elicited from him. His memory, which had been defective, was entirely lost, and his sleep was much disturbed so that he used to cry out and threaten vengeance on the neighbouring patients. Bed sores formed on his hips and sacrum, and they were with difficulty dressed and kept clean.

About the beginning of December, 1890, it was evident that he was much weaker and indeed worse in every way. He seemed to have considerable difficulty in swallowing, and his respirations at times were loud and stertorous. On the evening of the 14th of December he became comatose and died on the 15th.

Necropsy, December 17th, 1890.

Height $65\frac{1}{2}$ inches ; circumference at shoulders 44 inches ; *post mortem* lividity slight on dependent parts. A patch of reddish discolouration on right side of thorax due to an old burn.

Rigor mortis absent. General nourishment poor ; pupils dilated and equal in size.

Large bed sore over the sacrum ; on the left buttock was a slough $1\frac{1}{4}$ inches in diameter, and a sore over the right trochanter, 1 inch in diameter, partly cicatrised. Face and thighs jaundiced.

Heart weighed ten ounces ; *pericardial sac* contained one ounce of straw-coloured serum. Aortic valves .8 inches in diameter, mitral 1.2 inches, pulmonary 1.1 inches, tricuspid 1.3 inches.

Left lung weighed twelve ounces. Thirty ounces of straw-coloured fluid were found in left pleural cavity, and on the outer surface of both lobes was a layer of recent fibrinous lymph. Lower lobe in a state of congestion.

DESCRIPTION OF FIGURES, TO ILLUSTRATE PAPER
ON
"HYALINE DEGENERATION OF THE SPINAL CORD."

BY WILLIAM BULLOCH, M.B., C.M., ABERDEEN.

FIG. 1.—Section of the cord at the level of 4th cervical nerve, showing large cavity opening to the front by a wide opening and extending posteriorly and also into the right lateral half of the cord. The walls of the cavity are clear and hyaline, vessels dilated and extravasations to be seen in the neighbourhood; vessel walls hyaline and thickened. Grey matter is not distinguishable.

FIG. 2.—Section at the level of 5th cervical nerve. Cavity not so large and degeneration not so extensive as in Fig. 1. Vessels are all dilated and walls much thickened.

FIG. 3.—Section at the level of 7th cervical nerve. Hyaline degeneration chiefly in posterior columns, but also in lateral columns. All the vessels have thickened hyaline walls. No ganglion cells can be seen in grey matter. Central canal region occupied by a mass of hyaline. At the bottom of anterior fissure the artery of the fissure seen surrounded by hyaline material.

FIG. 4.—Section at the level of 5th dorsal nerve. Degeneration chiefly limited to posterior columns. Few cells to be seen in the grey substance. Hyaline mass in central region.

FIG. 5.—Section at the level of 12th dorsal nerve, showing degenerated tract limited chiefly to the postero-internal columns. Large mass of hyaline in region of central canal, with ill-defined cellular elements in its centre.

FIG. 6.—Section in the upper lumbar region. Central hyaline mass well defined. Degenerated tract in posterior column small.

[Autotyped from original pencil drawings, by Thévoz & Co., Geneva.]

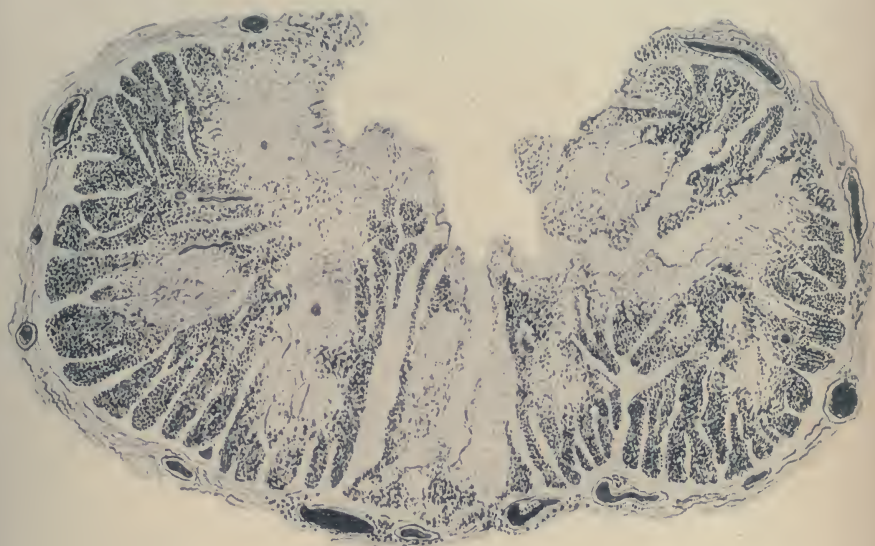


FIG. 1.



FIG. 2.

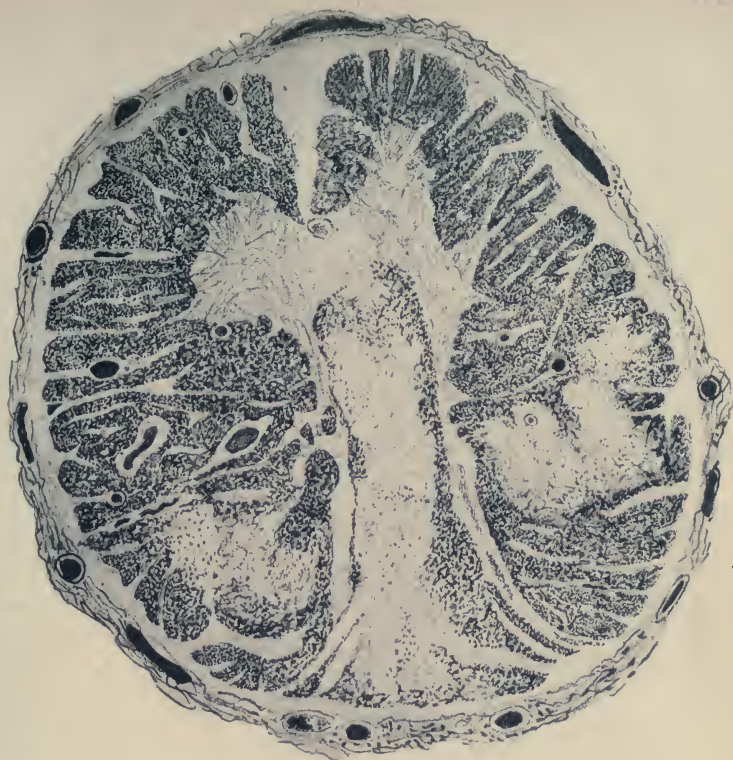


FIG. 3.



FIG. 4.

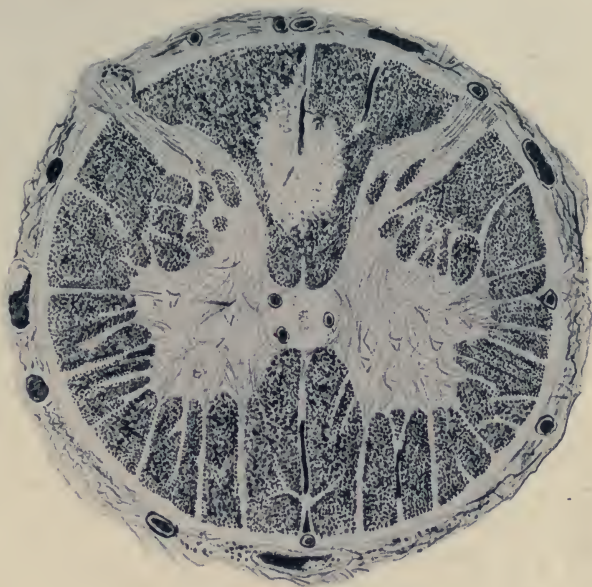


FIG. 5.

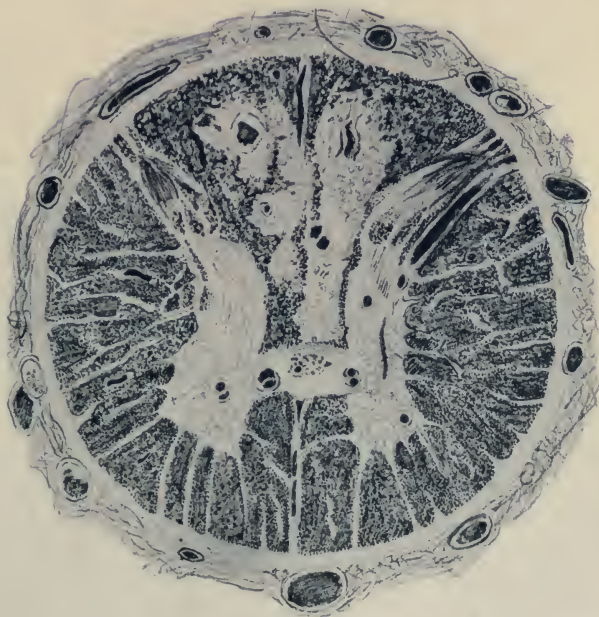


FIG. 6.

Right lung weighed 1lb. 5oz., and the pleural cavity contained ten ounces of serum. Old fibrous adhesions of pleura to diaphragm and costal parietes on the outer side. Lower lobe congested and œdematous.

Liver weighed 3lb. 11oz., and was congested and nutmeg-like in appearance. Gall bladder contained two ounces of bile.

Kidneys appeared to be normal, and were five ounces in weight respectively.

Spleen weighed nine ounces; consistence appeared to be softer than normal.

Nervous System.—One ounce of straw-coloured serum in subdural space of the brain. Dura mater adherent to vetex of brain, especially along the margin of longitudinal fissure. Pia cloudy over vertex. The brain presented nothing abnormal on the surface. The vertebral, basilar and middle cerebral arteries were slightly atheromatous.

Spinal cord.—Dura appeared healthy, but the cord itself was distinctly softer than normal, especially at a point extending from the third to the fifth cervical nerves. Here the contour of the cord was much altered, and the substance appeared to be diffuent, so that when cut into at this level a cavity was seen which at its lower part was more of the nature of a fissure extending deeply into the substance of the cord so as almost to divide it in two.

The *cauda equina* appeared healthy.

The entire brain, pons, medulla, cord and parts of the peripheral nerves were hardened in Müller's fluid and afterwards in Ehrlitzki's fluid preparatory to the Weigert hæmatoxylin process. After hardening, some of the pieces were dehydrated and embedded in celloidin. All the sections were cut in the ordinary freezing microtome. As regards the staining reagents, many were tried. Weigert's method and Pal's gave good results as far as showing the degenerated tracts, but the finer changes were seen best in specimens mounted in Färrant's medium or glycerine.

Osmic acid $\frac{1}{4}$ per cent. yielded splendid results, especially after colouring a little with $\frac{1}{4}$ per cent. watery solution of eosin or carmine.

Picrocarmine and logwood with eosin clearly demonstrated the nature of the changes, as did also many of the anilin colours, especially anilin blue, either alone or after treatment with eosin. Sahli's methylene blue method also gave good results.

Microscopical examination of nervous system.—In the cervical region, sections at the level of the broken down cavity showed that profound changes had taken place. The cavity opened by a wide aperture with rugged uneven edges towards the front. Below the cavity was more defined and presented more the appearance of a fissure. The anterior horns were fixed widely apart, and the cleft extended through the commissure into the posterior columns, which it traversed almost completely to the periphery. At one part a lateral branch from the cavity passed into the right lateral region of the cord through the grey matter in this situation.

Membranes.—The pia mater was enormously thickened and most of the blood vessels distended to their utmost with blood corpuscles, so as to give almost an appearance of cavernous sinuses. Many of the blood corpuscles could be seen lying in the walls of the vessels, and also in the neighbouring tissue beyond. Besides the distension of the vessels the walls were very much thickened, and of a homogeneous glassy-like appearance. The thickening was most apparent in the middle and outer coats, but the inner coat also participated in the change. At certain points the walls of the vessels had given way and allowed an extravasation of blood corpuscles into the neighbouring tissues. In most of the vessels masses of some clear, finely granular material could be seen, and the same appearance was frequent in the neighbourhood.

Round about the cord there could be seen a clear hyaline layer of the same material. This was evidently the layer of pia lying next the cord, which had undergone some transformation. On staining with eosine or carmine this layer was seen to be of a deep pink colour, while the rest of the pia was of a much lighter tint. The hyaline-like masses in the vessels were also stained very deeply.

This layer of deeply-stained tissue was continuous with

the septa, which were prolonged into the cord, and which also were deeply stained by carmine. The septa were much thicker than normal, and were composed of some transparent, finely granular material. In most of them enormously thickened and distended bloodvessels could be seen, and in the neighbourhood of these the hyaline material was present in large amount.

Cervical region.—On examining the substance of the cord it could be seen that the new hyaline element had been infiltrated between the elements of the cord and compressed the axis cylinders, leading to their degeneration. Between and around the axis cylinders all over the cord the hyaline could be seen in great abundance. In the posterior columns the hyaline had accumulated to an enormous extent, and large masses of it could be seen with broken-down axis cylinders in the neighbourhood. Here and there in the lateral columns, but having no systemic distribution, the hyaline material was in large patches, the axis cylinders having completely disappeared. In the grey matter, which was almost unrecognisable, the hyaline was abundant. No trace of any nerve cells could be seen.

The changes in the nerve fibres were seen in different stages. Where the hyaline was in small amount the fibres appeared irregular in outline, and there was no distinction between the axis cylinder and the myelin sheath. These stained deeply with osmic acid. Others were seen only lightly stained, and they were smaller in size and full of black granules, whilst the furthest stage appeared to be the conversion of the fibre into a shrivelled granular mass, which was surrounded completely with the hyaline material. Here and there in the degenerated tracts blood corpuscles could be seen which had exuded from the distended vessels.

At the level of the fifth cervical nerve the cavity was much smaller, and, in fact, was a split in the cord. The edges of the split were lined with hyaline. Descending the cord it was found that the changes were not so marked.

A section at the level of the seventh cervical nerve showed that the well-defined patches of degeneration which existed higher up in the lateral columns had disappeared. A few

healthy axis cylinders could likewise be seen in the posterior columns, although at certain places there were still large masses of perfectly hyaline stuff to be seen throughout the section. The commissural region was completely filled with the hyaline, which was continuous with a large hyaline mass lying at the bottom of the anterior median fissure around the artery of the fissure.

In the midst of the commissural hyaline mass the central arteries could be seen with thickened transparent walls, and full of blood corpuscles. Indistinct cellular elements could also be recognised here and there in the mass.

Dorsal region.—Here the changes were still less marked. The grey matter appeared more normal than in the upper regions of the cord, but still no healthy ganglion cells could be seen. They were converted into granular masses in which no nucleus could be seen. The changes in the commissure were similar to the above except that they were much better defined, so that the mass in this region looks like a minute tumour to the unaided eye. In the upper dorsal region the posterior columns were still widely degenerated except along the margin of the posterior nerve roots, where many healthy axis cylinders could be seen.

In the lower dorsal region the degeneration was still less marked. On each side of the posterior median septum next the periphery of the cord there was a triangular area, which was not infiltrated with hyaline.

Still further down the triangular area increased in size, so that the degenerated tract occupied a site corresponding to the distribution of the internal posterior and the postero-median arteries, but extending up to the commissure. The central mass is well defined, and in it, besides the hyaline, there can be seen numerous cellular elements.

In the upper *lumbar region* the degeneration in the posterior columns was limited to a small area on each side of the posterior median septum. In the middle of the hyaline degenerated area in several of the sections the distended and thickened postero-median artery could be seen. A few ganglion cells could be seen in the anterior cornua, but they were limited to the antero-lateral, postero-

lateral and central groups of cells, and in each of these groups the number of cells was much diminished compared with normal. The cells were likewise merely granular masses, which insensibly merged into the hyaline matter surrounding them.

The pia was still much thickened, and most of the septa enlarged and homogeneous. The mass in the central region was still well defined like a small tumour. The nerves of the cauda equina were also involved in the change, although no degeneration of the fibres had taken place. The vessels were all much thickened and distended with blood corpuscles, the septa between the fibres were also enlarged and homogeneous, and small quantities of the hyaline could be recognised throughout the bundles. At the tip of the spinal cord the canal was much widened and filled up with cellular elements, and also with the hyaline. The degeneration in the posterior columns became unrecognisable about the fourth lumbar nerve.

Sections of the peripheral nerves appeared to be normal. Medulla and pons also appeared healthy, and microscopic examination of the brain revealed nothing. Above the cavity in the cervical region the cord was not much altered, except that pia and some of the septal vessels were thickened. There was no ascending degeneration to be seen.

Summary.—The exact nature of the changes was by no means clear. The hyaline material had probably exuded from the blood vessels and had found its way among the nervous structures of the cord, surrounding them and leading to their destruction. The ganglion cells were converted into granular masses in some instances, while in others they were represented by hyaline masses the shape of the original ganglion cells. That the hyaline material was an exudation from the vessels seems apparent from the fact that it was in greatest amount in the neighbourhood of blood vessels, that the vessel walls were hyaline, and that hyaline masses were found inside the vessels.

In regard to the material itself nothing very definite was made out.

Histologically it was deeply stained by carmine, logwood,

and especially the anilin dyes. A differential stain, which showed the ramifications of the hyaline among the nervous structures to perfection, was obtained by staining the sections in a watery solution of anilin blue, and then washing them in a weak watery solution of eosin. The hyaline material all through the section was stained bright blue, the remaining structures being pink for the most part. The walls of the thickened vessels were blue, while the blood corpuscles in their interior were pink.

Anilin blue alone stained the hyaline of a deep blue colour, while the healthy parts were of a much lighter shade.

Gentian violet gave no differential colour reaction such as is obtained with amyloid. Fuchsin stained the hyaline very deeply. Iodine coloured it light yellow. In the central region the hyaline mass was extremely tough, so that it was with difficulty disintegrated by firm pressure on the cover glass.

Liquor potassæ broke it up into masses and caused it to swell. Acetic and sulphuric acids were without action upon it.

Conclusions.—Reviewing briefly the clinical condition, there was first a severe constitutional condition of anæmia, and when this seemed to be improving the paralytic symptoms set in, and they were of the type of an acute ascending paralysis. The paralysis began in the lower limbs and then involved the hands and arms, but there it stopped: preceding the paralysis there were the ataxic movements, showing a probable lesion of the posterior columns. We had here to deal with a compound lesion involving both the grey matter and the white. The wasting of the limbs apart from other considerations compelled the reconsideration of the diagnosis of Landry's paralysis, and it was then considered to be a case of "Subacute diffused atrophic paralysis" ("*paralysie générale spinale antérieure subaigue*," Duchenne).

The *post-mortem* changes in regard to their sight corresponded to the clinical symptoms. The ataxia was explained by the lesion of the posterior column, and the motor atrophic paralysis by the almost complete destruction of all the ganglion cells along the whole course of the cord.

In regard to the nature of the lesion, it is probable that the previous condition of the blood when the patient had been suffering from profound anæmia was the prime factor at fault. The composition may have been altered and some albuminoid product produced. This formed inside the vessels and then exuded through the walls, rendering the latter homogeneous and hyaline. The substance then accumulated round the vessels in the cord and pia, and finally gained the lymphatic sheaths from which it came to act directly on the nervous elements of the cord, cells, and axis cylinders, causing complete atrophy and degeneration.

The distribution of the hyaline material was peculiar. In the cervical cord it was in enormous quantity, and had broken down, forming a rugged cavity, while below it got gradually *less and less* in the *posterior* columns, differing altogether from an ascending degeneration. This is probably another proof of its distribution by the blood vessels. The central mass, which was so apparent, seems to have been derived from filling up of the canal by hyaline, which had exuded from the central arteries of the cord.

For much help in the explanation of the complicated changes which were present in the cord, and for the unstinted use of his laboratory appliances, I desire to tender my best thanks to Professor D. J. Hamilton, of Aberdeen.

TREMOR IN GRAVES' DISEASE.

BY A. MAUDE.

THIS important symptom was first discovered by Charcot in 1856,¹ in the earliest French record of a case of this disease: Trousseau also noted excessive tremor in one instance. It did not escape the observation of Sir Morell Mackenzie in 1868,² while Dr. Hector Mackenzie³ finds it noted in the records of St. Thomas' Hospital more than a dozen years ago.⁴

But on the whole this important factor in the recognition of the disease seems to have attracted little attention in England. Dr. Gowers' book is the only English text-book which mentions it, and in the discussion at the Ophthalmological Society in 1886, Dr. S. West, alone alluded to it.

Unquestionably it is a very constant symptom in Graves' disease, for I found it present in a typical form in six patients of a series of nine taken at random;⁵ only two years ago, however, a patient was shown at a provincial medical society, and the tremors were ascribed to alcoholism.

The symptom has been very fully studied in France, chiefly by Charcot⁶ and Marie, and the important monograph⁷ of the latter embodies most of our knowledge on the subject.

¹ *Gaz. Med. de Paris*, No. 38.

² *Trans. Clin. Soc.*, 1868.

³ *Lancet*, ii., 1890, 545.

⁴ Russell. *Med. Times & Gaz.*, 1876, p., 250.

⁵ A number of references in French literature are given by Marie. The more important are:—Chvostek, *Weiner Med. Presse*, 1870, Nos. 38, 39, 40, 42; Féréol, *Soc. Méd. des Hôp.*, Nov. 13, 1874; Delasiauve, *Soc. Méd. des Hôp.*, Nov. 27, 1874; Raynaud, *Thèse de Paris*, 1875; Rey., *Thèse de Paris*, 1877; Teissier, *Congrès de Clermont pour l'avancement des sciences*, 1876; Douglas, *New York Med. Journal*, 1879; Russell, *Med. Times & Gazette*, p. 250, 1876; Wynne Foot, *Dublin Journ. of Med. Sc.*, Nov., 1880; Nothnagel, *Wiener Allgemeine Zeit.*, p. 501, 1882.

St. Bartholomew's Reports, xxvii., 1891. Cases of Grave's disease, by A. Maude.

⁶ See *Leçons du Mardi*, 1888-9, p. 231 (et sequit.).

⁷ *Contributions à l'Etude et au diagnostic des formes frustes de la Maladie de Basedow*, Paris, 8vo.

I think it would not be too much to say that tremor is always present at some time or other in the course of the disease ; even when we fail to find it on examination we shall learn that it occurs on excitement, or on any sustained muscular movement. In cases that begin rather suddenly, and settle down into an ill-marked condition, it has generally existed early, and does not last very long ; but as a rule tremor is a late symptom. One patient of mine who had had goitre and exophthalmos for twenty years, had never noticed the tremor till a year before she came under my charge ; and in all my cases except two it has been long after the establishment of other cardinal signs that it appeared. In one instance the patient, after some years of futile treatment by drugs, has improved very greatly under the continued use of a Leiters tube to the epigastrium ; tremor has been present in a slight degree for a year or two, but since the general symptoms have improved the tremor has become far more persistent and marked.

The degree varies so much that in many cases the patient is quite unaware of it, and the condition is generally only noticed at first by the subjects of it because it interferes with delicate movements of the fingers, such as writing or sewing ; but it may become so severe as to cause her to drop cups and plates, as in my patient's case, or even to prevent walking,¹ or preclude the physician from feeling the pulse.²

My experience confirms the observation that the movements are as a rule very small in amplitude, strikingly regular, and are not dependent on voluntary motion.

The vibration is most marked in the hands, and may in very slightly marked cases be confined to them. The other muscles most affected are the pectorals, the serratus magnus and the erector spinæ. But if the tremor is well marked in any group of muscles it is sure to be general, over the whole body, to some degree. I have seen one lady who had never noticed it in her hands, where it was slight, but had noticed it in the muscles of the back ; it was confined to these two muscular groups.

Tremor is usually symmetrical, but not necessarily ; and

¹ Charcot.

² Russell.

has been known to be unilateral, when the goitre or exophthalmos are also unilateral.¹ In cases where it is well marked, the patient is in a state of perpetual vibration, sitting or standing. It is then much marked in repose, and only slightly exaggerated during movement.² But though muscular repose does not necessarily free the patient from tremor, mental repose does so to a great extent. It is always increased by agitation or "nervousness": it is produced or increased by hurried muscular action, or by any attempt to perform a movement carefully or accurately, especially if the patient is aware of her tremulous condition.

It is easy to ascertain the existence of even slight tremor by placing the hands on the patient's shoulders when she stands up. In this position the vibrations are, in well-marked cases, best felt in the vertical direction, but even in slight cases, where no vertical movement is felt, a small lateral oscillation can often be seen, if not felt.

On stripping the subject we may perceive that the muscles of the extremities and trunk are in a state of continual fibrillary movement, "that the whole surface of the body is the seat of a kind of general palpitation of a most curious character." (Marie.)

The tremor may be so exaggerated as to affect the respiratory muscles, producing a jerking laryngeal sound audible at some distance.³ To study this condition carefully, make the patient stretch out her arms, stripped, with the hands lightly extended. The whole limb is then seen to be affected by rapid contractions of the muscles, the amplitude of which varies in different subjects. The movement is usually fine, and is always quite regular and constant. The oscillations are most easily visible in the hands, and they affect the hand as a whole, being produced in the flexor and extensor groups of the forearm. The fingers have little individual movement, and the interossei especially have no share in it. The wrist is the joint most affected by the oscillations, and probably the fact that the flexion and extension movements are more marked than the supination

¹ Gowers.

² Féréol.

³ Charcot, *loc. cit.*

and pronation is due to the greater ease with which the wrist moves in the former plane, than to any election of groups of muscles in the forearm on the part of the tremor. The oscillations are more visible in the hands, not because the hands are farther from the point of support of the limb, but because the muscles of the forearm are affected most.

This "flexor tremor" is also characteristic of alcoholism, but at the same time in alcoholism there is distinctly more contraction of the interossei and consequent lateral deviation of the fingers.

In the same way the movements of the lower limbs may be studied. Let the patient stand with the foot extended, just pressing the floor with the toes; the vibrations are then usually marked. Then let her sit with the knees at a right angle, and the toes touching the floor, and we shall find in a well-marked instance, the same sort of vibration as in "ankle-clonus" (Fuss phænomen). In this connection I examined the patellar reflexes in five cases, and found them absent in four, and feeble in the fifth. Tremor is usually constant in any one patient for a long period, but it has terms of exacerbation, just as have all the other symptoms; so that in many cases the habitual or constant tremor passes unnoticed by the patient, but the exacerbations are observed.

I have not succeeded in finding any direct relation between the increase of tremor and increase of other symptoms.

Marie has obtained mechanical tracings of this tremor, and gives us an excellent account of them in his monograph; and to his work I am entirely indebted for knowledge on this part of the subject.

Registration of the vibrations was obtained by two methods. First, by a modification of Marey's tambour, applied to the dorsal aspect of the metacarpus; secondly, by means of an india-rubber ball or "pear" grasped in the patient's fist (like an enema syringe) not tightly enough to compress it. Of course, in both cases the tambour and "pear" are connected with an ordinary recording apparatus, furnished with a tuning fork to record the rate.

Marie and Boudet also tried to obtain tracings by means of Rosapelly's apparatus; but the instrument only gave the frequency of the vibrations, without any record of their amplitude.

In all comparative experiments of course the same mechanism, pressure and tuning fork must be used.

From the tracings we learn, first as to the *rapidity* of the oscillations, that they are strikingly regular in rhythm; the interval between the crests of the successive waves varies very little; in fact the tracings are almost as regular as those of a tuning fork.

In eleven cases, in which tracings were taken by Marie, the rate of movement was almost the same in all, varying only from $9\frac{1}{2}$ to 8 oscillations per second, with a mean for all cases of $8\frac{1}{2}$. As to amplitude, in the vertical direction the oscillations present anything but regularity. If we examine tracing 1 of Marie, (p. 20), we see that the waves increase gradually for a certain number and decrease again. The tracing presents a series of roughly fusiform enlargements, described as nodes (nœuds) by M. Fernet in his monograph.

These variations in amplitude are very irregular, and no explanation has yet been offered as to their causation, but they certainly have no correlation with the movements of respiration or circulation. A distinct correlation is however found to exist between the increased waves and the occurrence of cough; an attack of cough produces a greater amplitude of vibration without disturbing the rate. This change is probably merely due to the shock of the chest wall, produced by the cough, being transmitted through the shoulders and arm to the hands.

In reply to the question whether there is any direct relation between the degree of tremor and the intensity of other symptoms, my own impression is strong that there is none. Not only, as I have said above, may it become most pronounced as other symptoms are improving, but it is more constant, in individuals who present it, than any of the other (notoriously inconstant) symptoms of the disease.

It has also no relation to a general emotional (or "nervous") state, as hysterical tremor often has.

When we compare the tracings produced in Graves' disease with those produced by other forms of tremor, the difference is instructive. Such other forms are senile tremor, paralysis agitans, general paralysis, disseminated sclerosis, hysteria, and poisoning by alcohol, mercury, lead or arsenic.

The tracings obtained in Graves' disease are exactly

similar in time and form to those produced in animals after excision of the thyroid gland.¹

Mr. Horsley also drew attention to the fact that the tremor wave in paralysis agitans is a compound wave, a summation of two separate waves, as the larger convulsive tremor waves in myxœdema are summations of smaller ones.

The correspondence of the tremors in myxœdema and exophthalmic goitre afford some evidence of the seat of production of the tremor; for Schiff had shown that the tremor in cachexia strumipriva was of central origin, and Horsley confirmed the observation, while he carried the investigation further, for he removed in one case the centre in the cortex for the upper limb; the result was complete brachiooplegia, but no interruption of the tremors; thus proving that the tremor originated not in the cortex but in some lower centre, either in the basal ganglia, the bulb, or the spinal cord; and support is given to this assumption by the fact that at first voluntary effort abolishes the tremor, that the inhalation of ether increases it, and that it disappears on reflex irritation.

Moreover the rate of vibration is 8 to 10 per second at first, and later some multiple of these numbers, which is the rate determined by Loven for the spinal cord in frogs, and probably also the rate for the spinal cord in man (Horsley).

Mr. Horsley, however, admits that none of these experiments disprove the possibility of the tremors being idiomuscular.

In the disease we are studying we have seen the rate of vibration is very constant, 8 or 9 per second; in senile tremor it is much more slow, $4\frac{1}{2}$ to $5\frac{1}{2}$ (Marie); in no case above 6 per second. In paralysis agitans the rate varies considerably in different cases; it may simulate disseminated sclerosis in its slow rate and its irregularity. Charcot gives an average of 3 to 6 oscillations per second, and Marie an average of 5. On the other hand it is quite exceptional for the movements of insular sclerosis to be as fine and regular as in Graves' disease.

The movement in Graves' disease and mercurial poisoning are shown by Charcot² to differ only in rate, and in the fact that in the former affection they are much more continuous and regular, and are not increased, as a rule, by intentional muscular action.

In general paralysis and alcoholism the rate of vibration may be much the same as in Graves' disease, but tracings

¹ Brown Lectures, 1885.

² *Leçons du Mardi*, 1, p. 378.

show that while the general line in the latter is regular and straight, that produced in the other two affections is much more wavy and jerky. Moreover in Graves' disease the intrinsic muscles of the hand and tongue escape, while they are particularly affected by alcohol poisoning.

Tremor in lead poisoning is so rare and varies so much in character that it is impossible, as yet, to draw distinctions on this head.

An important feature which separates that of Graves' disease from tremor due to metallic poisoning is the entire escape of the muscles of the head, neck, lips and tongue.

I have not sought to press the diagnosis of this form of tremor from others, but merely by indicating what I believe to be its salient features, to leave to the cultivated neurologist its differentiation from other forms better known.

The affinities of this tremor are rather to purely "asthenic" trembling, such as follows in many subjects an acute illness, or to the tremor of old age.

From hysterical it is separated broadly by its great regularity in rhythm, but, of course, so-called hysterical trembling may resemble that of Graves' disease, as it will occasionally imitate that of any other neurosis.

The points on which I would lay stress, are :—

(a) The great frequency of it in cases of exophthalmic goitre.

(b) Its constancy when it does exist.

(c) Its constant character.

It may be observed that I make no mention of chorea in this neurosis. The condition has been noted by observers fully capable of differentiating true choreic movements, but there is no evidence as to whether it exists or not side by side with the ordinary trembling.

We ought not to lay too much stress, except for diagnostic purposes, on differences in tremor in various diseases. We know nothing about the pathogeny of tremor, or of the seat of the lesion, and it may well be that what appear to be distinct forms of trembling owe their difference to the fact that certain muscles are affected rather than to the fact of different causation.

REMARKS ON THE PATHOLOGY OF SO-CALLED PACHYMEINGITIS INTERNA HÆMORRHAGICA.¹

BY J. WIGLESWORTH, M.D.LOND., M.R.C.P.

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IT would be foreign to my purpose to-night to enter upon a detailed account of the affection commonly known as *Pachymeningitis interna hæmorrhagica*, my object rather being to offer a few remarks in favour of the hæmorrhagic origin of the affection, in opposition to the view which ascribes the phenomena met with to an inflammatory process, which latter view, if we are to judge from descriptions given in text-books, appears to be still the prevailing one.

As is well-known the hæmorrhagic theory is not a new one; it is in fact the older one of the two, and was advocated by Prescott Hewett and others, before Virchow lent the weight of his authority to the inflammatory doctrine, and it is mainly to the writings of that eminent pathologist that we are indebted for the predominance of the inflammatory theory at one time almost universally accepted.

It is unnecessary for me to quote the usual description given in text-books when treating of this affection; the formation of an inflammatory film upon the inner surface of the dura mater—the organization of this—the formation of similar films superimposed one over the other—the rupture of newly-formed vessels—and so on. All these descriptions start with the assumption that the primary thing is an inflammatory exudation upon the inner surface

¹ Read at a meeting of the Neurological Society, May, 1892.

of the dura-mater, and that the hæmorrhagic element met with is a secondary result of the rupture of newly-formed vessels in this inflammatory membrane.

I take the exactly opposite view to this, and contend that the membranes found adherent to the inner surface of the dura-mater, be they simple or laminated, small or large, filmy or thick, hæmorrhagic or fibrinous, are all the direct result of hæmorrhagic effusions into the sub-dural space (arachnoid cavity); and that any inflammatory changes which may be met with in the dura-mater, are purely secondary and due to the irritation set up by the effused blood.

I may briefly sum up the chief arguments in favour of this view as follows:—

If the membrane were the primary thing, and the hæmorrhage secondary, although we might get the membrane without the blood, we could not get the blood without the membrane.

But as a matter of fact cases are continually met with—I have seen many such—in which effusions of blood are found in the sub-dural space without the presence of any trace of membrane upon the inner aspect of the dura-mater. Sometimes the effusion is a copious one, at other times a little recent blood or clot is found smeared over the upper surface of the arachnoid, or a recent clot may be met with loosely adherent to the inner surface of the dura, just stuck on as it were; or all these conditions may be combined. Of course it may be argued that these cases should not be included in the same category as those in which a distinct membrane is present; but the two things occur under such exactly similar sets of conditions, and there is such a very gradual transition from one to the other, that the conclusion seems forced upon us that they are merely stages in the same pathological process.

Again, if the membrane were the result of a primary inflammatory exudation from the vessels of the dura-mater we should surely find some evidence of inflammatory changes in this structure itself. But, as a matter of fact, such changes are for the most part conspicuous by their absence.

This is so at least in all quite recent cases. If we detach the membrane from the inner surface of the dura-mater, to which it is always but loosely adherent, we find the endothelial surface of this latter membrane smooth and shining and exhibiting no sign of departure from health. There is no thickening, no roughening, no undue vascularity. Later on, as the new membrane tends to become organized, vascular adhesions form between it and the dura-mater, and on the separation of these, which can always be readily effected, the inner surface of the dura may be found a little roughened and at times a little injected, and the dura itself may perhaps be a little thickened. But, I repeat, even these changes, slight though they be, are not found in the recent cases, and I regard them purely as secondary results of the irritation set up by the effused blood, and as quite comparable to the changes which take place in the walls of a vessel within which coagulation has occurred.

The structure of the newly formed membrane itself is entirely in harmony with the supposition of its hæmorrhagic origin; for if recent it is found to consist of a meshwork of fibrin in which are entangled red and white corpuscles; whilst in the older cases, when capillary vessels have been formed in it, bands of imperfectly formed fibrous tissue may be seen running parallel with one another containing in their interstices collections of red blood globules, or it may be simply the remains of these in the form of little heaps of hæmatoidin granules; the whole in fact having much the appearance of an organizing or organized thrombus.

The age at which this affection is most frequently met with is distinctly that of advancing life with its concomitant arterial degeneration and tendency to hæmorrhage. Out of fifty-four cases observed by myself, which I have analysed with reference to this point, the average age was 51.61; the mean age of the asylum population from which the cases were drawn being about 43. When occurring in young persons it is almost always in association with general paralysis, in which disease it is common to meet with degenerative changes in the small vessels of the pia-mater and cortex cerebri.

It is worth noting that the affection is by no means always bilateral, although it is so in the majority of cases. Thus out of fifty-four cases, twenty were entirely unilateral and thirty-four bilateral. One would have anticipated that had the affection been an inflammatory one it would have affected more uniformly both sides of the cranium.

The rarity with which the affection occurs in the cerebellar fossæ is also of interest. Whilst it is not uncommon to meet with a hæmorrhagic membrane on the upper surface of the tentorium cerebelli, it is very rare to meet with one beneath it. I do not think I have ever seen an instance of it. On the inflammatory theory this is difficult to account for; but the mechanical obstacle which the tentorium must present to the gravitation of blood into this region, supplies a ready explanation of its exemption.

The conditions under which this so-called pachymeningitis is met with are of importance in determining its pathology. Unquestionably the immense majority of cases are met with amongst the insane, and hence it is chiefly in asylum practice that it comes under notice. Next in frequency it occurs in the subjects of chronic alcoholism, a condition indeed closely allied to insanity. Its occurrence under other circumstances appears to be very uncommon, if we are to judge from published records; and on this point I am anxious to hear the experience of others. Furthermore, it is not in the recent acute cases of insanity that it is commonly found; although it occasionally so occurs, it is nevertheless comparatively rarely met with except when the insanity has been of some duration. General paralysis is the disease in which it most commonly occurs, about half the total cases of pachymeningitis having this association; but no form of insanity is exempt, the different forms of dementia furnishing a good proportion. In other words the affection is associated in overwhelming proportion with *brain degeneration and atrophy*. And this leads me to what I consider to be the main factor in the production of the disease, namely, the loss of support experienced by the meningeal vessels in the degenerating and wasting brain, assisted no doubt by the localised or general congestions of

the meninges which are so liable to occur in all forms of insanity, especially in general paralysis. Probably the source of the hæmorrhage varies in different cases; it unquestionably at times comes from the small vessels of the pia-mater occupying the summits of the gyri, for I have seen cases in which a recent clot, or a hæmorrhagic membrane, has been associated with a small sub-arachnoid hæmorrhage in this situation, and have been able to trace the rupture in the arachnoid through which the blood has been effused into the subdural space. That the blood does not diffuse itself more extensively throughout the subarachnoid space is doubtless due to the fact that in many cases of insanity, especially in general paralysis, there is a more or less intimate union between the pia-mater and arachnoid, so that in bursting through this latter membrane into the subdural space the blood would be simply following the lines of least resistance.

A further fact of some significance is that as a rule the affection does not declare itself by symptoms, the pachymeningitic membrane being discovered after death in cases in which its presence had not been suspected during life. Doubtless this is in part, perhaps mainly, due to the fact that the affection usually occurs in insane persons in whom symptoms of all kinds are masked. But even demented patients are liable to be disturbed by affections producing brain irritation and pressure, and I cannot but think that the comparative rarity of symptoms is another argument in favour of the compensatory character of the affection, the inference being that in the majority of cases the effused blood does little or no more than fill up the space left by the wasting brain. In a minority of cases, however, the hæmorrhagic effusion, once started, is not so readily limited, but becoming copious produces signs of irritation and pressure, declaring itself by such symptoms as convulsions, paralysis, &c. I could narrate several such cases, in some of which a copious effusion proved rapidly fatal, whilst in others, the patients having lived a variable time after the onset of symptoms indicative of such an effusion, there was found after death a hæmorrhagic or fibrinous membrane adherent to the under surface of the dura-mater.

A word or two with respect to the lamination which many of these pachymeningitic membranes present. Doubtless the appearance of successive layers has had much to do with the notion of an inflammatory process, but I fail to see how this condition favours the idea of inflammation as opposed to hæmorrhage. I think that without doubt this laminated appearance is due at times to fresh hæmorrhagic films being superimposed upon those of older date which have already become more or less organized, the same conditions which produced the first effusion continuing in operation. But I have little doubt that a single large hæmorrhage is capable of producing in time a laminated membrane owing to the changes which take place in the clot. One of the specimens exhibited from a case already published illustrates this. Here the hæmorrhagic membrane which was clearly occasioned by a copious hæmorrhage seven days before death, showed when fresh a dark centre bounded by pale lines and a tendency for the fibrin to split into layers parallel to the surface—clearly an incipient lamination. It is not, however, to be denied that rupture of newly-formed vessels, in an organizing membrane of this description occurs at times, producing fresh hæmorrhages. I show a figure and drawing of such a case in which a small recent clot is seen lying between two thin layers of membrane, producing when fresh something of the appearance of a cyst—hence one of the names, “Arachnoid Cyst,” under which this affection has been described. The hæmorrhages thus produced, however, appear to be mostly of very small dimensions.

In bringing before you these few facts and arguments in favour of the hæmorrhagic origin of the affection under consideration I do not wish to be understood to deny the existence of a true cerebral pachymeningitis.

All I wish to contend for is, that the condition which usually passes under that name has no right to be called by such a title; and that it would be better to discard it altogether, and adopt one more in accordance with pathological facts.

Clinical Cases.

A CASE ILLUSTRATING KINÆSTHESIS.¹

BY WILLIAM B. RANSOM, M.A., M.D., M.R.C.P.

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THE following case of epilepsy, treated by trephining, afforded opportunities for observations and experiments such as have hitherto been few in man, and gave information on the subjective side which cannot be obtained from experiments on animals. Although therefore in some respects the results are less rigorously accurate than those of the physiological laboratory, yet I venture to think they shed some light on the mode of action of those areas of the cerebral cortex associated with voluntary motion, which by a majority of physiologists have been called simply "motor centres," but which Dr. Bastian considers to act more on the sensory side of the series of cerebral processes, and has called "Kinæsthetic."

It would be superfluous for me here to set forth the opposing arguments of the two schools, and I shall simply record briefly the aspects of this case which bear upon the problem.

History of Case.—T. F., a young man of 19, came into hospital under my care in April, 1891, complaining of fits.

Six years previously he had been knocked down and rendered momentarily unconscious. He walked home, however, apparently not much the worse, but next night had his first fit. During the next nine months the fits gradually increased in number and severity. Each fit began with tingling in the left thumb, which

¹ Paper read August 2nd, 1892, at the International Congress for Experimental Psychology, London.

was then the seat of spasm, which spread up the arm to the shoulder and face. He then usually lost consciousness. Occasionally he seems to have had the tingling sensation without subsequent spasm.

A year after the accident he had a very serious illness, probably meningitis, which left him blind. He recovered from this, but the fits continued, though with less severity.

A curious point is that about a year after this illness he began sometimes to have a visual aura preceding that in the thumb. This consisted in seeing "queer objects" and bright lights.

Condition in April, 1891.—Patient is a well-grown young man, with no motor defect except in the left hand. General sensation is normal except there. The sense of smell is absent, and he can at most perceive the difference between a dark room and bright sunlight. The optic discs are atrophied. There is no depression or scar on the head.

Left Hand.—There is some weakness of the muscles of the left hand: right grip = 160, left = 75. No wasting of muscles.

Tactile sensation is dulled below the wrist, particularly on the thumb, and the power of localisation is bad, two points being merged into one at a much greater distance than on the right side. There is partial analgesia. The muscular sense is markedly deficient. He fails to make out the position of his fingers or thumb, or to distinguish weights which are readily known as different by the right hand.

He cannot read his raised type with the left hand. All these deficiencies are more marked in the thumb.

Operation.—As treatment by bromides failed to relieve, I asked my colleague, Mr. A. R. Anderson, to operate.¹ This he did by trephining at a point $2\frac{1}{2}$ inches down the right rolandic line. Subsequently another disc of bone was removed, and a surface of brain about 2 inches by 1 inch exposed. A circular patch of this area, about 1 inch in diameter, was found to be depressed by a cystic accumulation of fluid between the pia and the thickened dura mater. Moderate faradization of this depression caused flexion of thumb and fingers; when the current was increased the contraction spread up the arm to shoulder, left side of face and left leg, and then a general fit ensued. The application of the electrodes to the healthy brain at the lower margin of the depressed area caused twitching of the left side of the face; when applied above the depression the left shoulder contracted.

¹ Mr. Anderson will publish the surgical aspects of the case elsewhere.

I merely mention these details to show the topographical agreement between man and monkey.

The thickened dura mater having been cut away, the bone discs were replaced and the wound closed. The patient made an excellent recovery. For a time he had fewer fits, but eventually they recurred and he began to have considerable pain in his head. It was accordingly decided to remove the discs of bone, and this Mr. Anderson did on March 29th, 1892, removing at the same time adhesions to the cortex. A moderate faradic current applied at the operation to the centre of the depressed area caused contraction of the thumb only. The patient again recovered readily, and left the hospital a month after the operation. Since then he has had no pain, very few fits, and those slight ones. I have carefully watched his progress, and have been able, with his full consent, to study the effect of faradizing the cortex through the gap in the bone without the sensorium being obscured by an anæsthetic.

The following method was adopted :—

The skin having been shaved and cleansed antiseptically, is dried with alcohol, and a little cocain is injected into the scalp to prevent the excitation of the cutaneous nerves becoming too painful. (The cocain may be omitted, but the skin sensations are, in that case, apt to be unpleasant.) The electrodes, two strong sharp needles, mounted at a distance of $\frac{1}{2}$ inch apart, are then thrust through the scalp into the trephine hole for a depth of half-an-inch.

The first onset of even a weak current may be somewhat unpleasant to the skin, but this soon wears off, and even strong currents can be borne.

As the electrodes at a depth of half-an-inch are probably not in actual contact with the cortex, moderately strong currents are needed to get definite results, but that diffusion does not take place to a wide extent is proved by the fact that the insertion of the electrodes in different parts of the scalp over the trephine hole caused distinct groups of muscles to contract. Thus movements of the elbow were caused when the needles were in the upper part, of the fingers when lower down; and I succeeded once in getting flexion of the fingers when the needles were in one part of the hole, and extension when they were removed half-an-inch away.

The following changes have occurred in the hand since the first operation. Motor power is somewhat better than before, but still rather weak; there is no analgesia, and tactile sensation is

distinctly improved, being now nearly as acute as on the other side, except on the ring and little finger. Muscular sense is similarly better, being also most deficient on the ring and little fingers. (It is curious that whereas before the operation the thumb was distinctly worse than the fingers, it has recovered to a larger extent.)

In making an experiment, tactile sensation, the muscular sense and motor power were tested before and after stimulation, and in some cases muscular sense was tested during the stimulation.

The following results were obtained :—

(1) It was possible, as already mentioned, by this method to cause contraction of different groups of muscles of the arm and hand.

(2) The first effect of a moderate current was to produce a *tingling sensation* in the part which would contract when the strength of current was increased. After repeated excitations with a given strength of current the contraction might cease to be produced, and only the sensation occur. The sensation might outlast the stimulus.

(3) After a strong induced contraction, voluntary motor power was weakened.

(4) While muscles were contracting under the influence of the current, this muscular sense was improved, and this improvement lasted a few minutes after the application of the current.

(5) No effect was observed on tactile sensation.

The above are the results which bear chiefly on the doctrine of kinæsthesia, but certain other points of interest may also be mentioned :—

The contractions caused by such stimulation might be limited to one finger or the thumb, or might affect a large group of muscles.

Thus in one experiment the first effect was a sensation in the fingers, then tonic closing of fist, then tonic flexion of elbow, and then clonic spasm of the whole arm. The spasm did not spread, and consciousness was not lost.

In another experiment the same result occurred, but in this, although the needles were removed when the spasm reached the shoulder, it spread to the leg and face, which

latter became congested. He never lost consciousness, but was able to get up off the chair and lie on the floor as a precautionary measure. He said "that was just like a real fit." As a rule, however, only moderate contractions, strictly limited to a few muscles, were obtained, so that it is probable that in the above two instances the hyperæsthetic epileptogenous focus was excited.

Another very remarkable phenomenon sometimes occurring was that during stimulation with quite a moderate current he would see a light, blue or white, mostly in the left eye, just as he frequently has done at the beginning of a fit. Possibly this visual aura and visual sensation produced by excitation of the arm centre may be connected with some pathological condition produced by the serious illness in which he went blind six years ago.

Great caution is needful in drawing conclusions from these observations, but I venture to think they go to show:—

(a) That depression of the activity of the cortical so-called "motor" centres is associated with the following changes in the corresponding limb or part of limb:—

- (1) Diminution of motor power.
- (2) „ muscular sense.
- (3) „ to a less extent of general sensation.

(b) That exaltation of their activity, even when artificially produced by the interrupted current, leads to the following changes in the corresponding limb:—

- (1) A vague sensation.
- (2) Increase of muscular sense.
- (3) Muscular contraction.

These conclusions I further think are not readily explicable on the theory of motor centres with which Dr. Ferrier has especially associated his name, but indicate a close connection with the afferent as well as the efferent series of cerebral processes, such as is postulated in the Kinæsthetic theory of Dr. Bastian. It would perhaps be fortunate if we could drop the term "centre" altogether, inasmuch as it implies the idea of an end-organ, remembering that whether viewed from the physical or psychical

aspects, vital actions are really reactions with a complicating time factor, and that to look upon these shunting places of nervous currents from the ingoing or outgoing side alone must always obscure half the truth.

NOTE.—At the meeting at which this paper was read Prof. Schäfer made the perfectly valid criticism that the sensation produced in the hand as the first result of electrically stimulating the “motor area” of the cortex might be due to vaso-motor changes in the hand, which changes were imperceptible to the observer. I am quite aware that it is impossible to prove the non-existence of unperceived processes (such as vascular changes or small muscular contractions), due to efferent impulses going from cortex to hand, which might be the cause of the sensation; but we cannot thus account for the complementary fact, viz.—diminution in tactile and muscular sense during depression of the activity of the cortical area.

TUMOUR OF CENTRUM OVALE, CAUSING PARALYSIS OF MOTION AND MUSCULAR SENSE; OPERATION; DEATH.

BY LANDON CARTER GRAY, M.D., NEW YORK.

I DO not deem it necessary to recapitulate all that has been written of late years concerning localization of the muscular sense, or to more than refer to the compilations by Dana and others. I think that the following case is really the first record, in the happy precision of the localisation and the short duration of the symptoms. It seems to me to settle beyond a doubt the question that lesion of the ascending parietal at least, can cause a loss of the muscular sense without impairment of the other senses.

On November 1st, 1890, Dr. I. H. White, of Richmond, Va., brought to my office a gentleman aged thirty-eight years, of whom the following history was given :—

Some fourteen days before, his right arm and leg had gradually become weak, the onset having been first in the foot, and then, a few days afterwards, in the arm. For about the same time he had suffered from headaches, worse in the morning, at first at the vertex, occasionally in the left side of the head, and on the morning of my examination across the brow. He had also, for about the same length of time, suffered slightly from insomnia, so that he could obtain only about four to five hours' sleep. Several years before this patient had what Dr. White supposes was Menière's disease. Some time before my examination an angioma had been removed by the doctor from the region of the knee by means of caustics. About a year before a hard growth had appeared in Scarpa's triangle, and now occupies the whole of this anatomical area. There has been great domestic trouble, about which the patient has been much worried. The patient has always stuttered, and this speech-defect does not now seem to be worse, and he has had no other defect of speech than this. There was a gonorrhœa twenty years ago, and following it possibly, Dr.

White thinks, a slight stricture. There has never been any discharge of pus from the ear, but there would seem to have been, at the time of the Menière's disease, some acute aural trouble, either of the labyrinth or the middle ear, and there is now deafness upon this side. There is no rheumatic or cardiac trouble, and there has never been any convulsion or loss of consciousness. The iodide had been administered in doses of fifteen to twenty grains, but invariably disagreed so greatly with the patient's stomach that it could not be pushed higher, or, indeed, continued.

Upon examining the patient myself, I found that there was a considerable motor paralysis of the right leg, so that the patient walked with great difficulty, and that there was also a slight motor paralysis of the right upper extremity, so that the hand-grasp was distinctly more feeble than upon the other side. In the foot the sensations of tact and pain were very slightly impaired, whilst the sensations of temperature were perfect. Tact was tested by means of the æsthesiometer, and by the differences between a rough cloth and the finger, and by the perception of objects held in the hand. The sense of pain was tested by means of a needle. The sensation of temperature was tested by means of three tumblers, one containing hot, one lukewarm, and the other cold water, and the patient promptly detected the difference in these by touching the outside of the glass with his fingers. But the muscular sense was so distinctly impaired that the patient was unable to tell which finger I took hold of, or what I did with it, or to tell me the nature of the muscular movements which I imprinted upon the foot as a whole. In the right hand the sensations of tact, pain, temperature, and the muscular sense were absolutely unimpaired. The cremaster reflex was very sluggish on both sides. The tongue pointed straight. The naso-labial folds were strong and equal. The right knee-jerk was slightly exaggerated and somewhat spasmodic, whilst on the left it was normal. I could detect no mental impairment whatsoever, although I ranged over a wide field of discussion involving judgment, perception, memory and temper. There was great deafness of one ear, seemingly due to an old otitis media. Careful ophthalmoscopic examination disclosed nothing abnormal about the discs. A detailed examination of the muscles of the eyes, eyelids, abdomen, and of all the muscles of the opposite side of the body, of the abdominal reflexes, and the tendon reflexes of the upper extremities, of the other ear, of taste, smell, and sight, disclosed nothing abnormal. I made a diagnosis of a tumour of the left centrum ovale. I believed there was a tumour because of

the presence of one in Scarpa's triangle, as well as because of the history of the precedent angioma. I believed that the tumour was subcortical because of the lack of a convulsion, together with the absolute mental integrity. I stated to the patient that I thought his paralysis would soon involve the arm and progress in the leg, advised him to go home and settle his affairs, advised his physician to administer as large doses of iodide and mercury as the patient could stand, and then, if the disease progressed, to have the patient trephined.

On November 13th the patient was again brought from Richmond by Dr. White. His leg was now completely paralysed, as was also the upper extremity, so that the patient could only make a slight movement of the fingers. The sensations of tact, pain, temperature, and the muscular sense were impaired to the same extent that they had been in the examination thirteen days before. In the right hand, however, the sensations of tact and pain had now become very slightly impaired, but the latter was not at all retarded; the temperature sensations were perfect, but the muscular sense was distinctly impaired, though not greatly so, the patient being unable to tell as to which one of the fingers was touched, or what movements were impressed upon any one of them, although he could speak decidedly when the whole hand or the arm or forearm were moved. The tongue still pointed straight, and the naso-labial folds were still strong and equal. The cremaster reflex was still very sluggish. In all other essential details, however, the patient was unaltered, except that his headache had now become intense, and he complained bitterly of it. This headache was not now confined to any particular portion of the head, although he seemed to think that it was greater over the brow. Although the patient was very somnolent, his intelligence was perfect, so that he put and answered questions with perfect intelligence, although slowly, and he gave me an accurate account of how he had willed his property and otherwise disposed of his affairs. In the last few days he had complained of roaring noises in the deaf ear. Since I last saw him he had had no convulsions and no vomiting. The left eyeball seemed slightly more protruberant than the right, but there was no ptosis nor strabismus.

On November 14th, at 2 p.m., he was operated on by Dr. John A. Wyeth; Drs. I. H. White, E. F. Ayres, J. F. Wynn, and myself being present. The skull was unusually eburnated, and it took an unusual time to cut through it. Sufficient of it was removed to lay bare the ascending frontal, the ascending parietal,

the bases of the second and third frontal, and about the anterior third of the first and second parietal convolutions. The dura bulged and was very tense, although it seemed to be normal. The pia was normal and not at all injected. When the membranes had been removed, the cerebral substance bulged markedly. As careful an examination was made of the cortex and subjacent white matter as it was possible to do by means of palpation and puncturing with a long needle, and this needle was thrust as far down as the ventricles in several different places. But no sign of a neoplasm was felt or seen. The patient stood the operation remarkably well, but upon recovering from the effects of the ether remained comatose, except for a few moments, when he said that his headache was gone. The temperature ranged at about 100° until about an hour before death, when it rose to 108°. He died the morning of November 17th. The autopsy was made about twelve hours after death by Dr. Dawbarn, there being present Drs. John A. Wyeth, E. A. Ayres, E. G. Mason, W. B. Pritchard, and myself. A slight pachymeningitis, evidently of long standing, was found over the paracentral lobule, but the pia and cortical substance were seemingly healthy. The calvarium was found to be unusually thick, and so marked was this thickness at the apex of the petrous portion of the temporal bone as to constitute a hyperostosis, most noticeable on the right side. There was no periosteal adhesions of the dura. At the site of the operation there were inflammatory changes, although these were slight. There were numerous *puncta hæmorrhagica* in the white substance, corresponding to the punctures that had been made during the operation. The cerebrum and the cerebellum were removed from the skull, placed upon a flat dish, and sections of the cerebrum were made from side to side vertically across the cerebrum, each of these extending in depth from the cortex through to the base. The first section was made about half a centimetre in front of the mid-point of the upper or first frontal convolution; the second section was made midway between the first section and the anterior border of the ascending frontal convolution; the third section was at the anterior border of the ascending frontal convolution, and the fourth section was at the posterior border of the ascending parietal convolution. Other sections were made behind this last, of course, of which it would be irrelevant to speak. Even by these different sections no neoplasm was discovered. I then proceeded to minutely examine each one of these sections, first by palpation, and then by the scalpel. In the first three sections nothing was discovered by these methods of examination.

In the fourth section nothing was detected by palpation, but very slight manipulation with the handle of the scalpel disclosed, close to the posterior border of the fourth section, a neoplasm about the size of a hickory nut and so soft in consistence that it was not distinguishable by palpation from the surrounding cerebral substance. It was found to have a cystic degeneration in the centre, and around the tumour there was no microscopical evidence of cellular changes in the white matter. *It lay, as will be seen, in the ascending parietal convolution, rather nearer the posterior border of this convolution, and was situated about one quarter of an inch beneath the surface of the convolution, at about the junction of the upper and middle third.* Upon section it was found that the tumour had been penetrated by one of the exploratory needles. A microscopical examination, kindly made by Dr. Beach, showed that this neoplasm was a round-celled sarcoma of the melanotic variety.

ANOTHER CASE OF ANÆSTHESIA DUE TO LESION OF THE GYRUS FORNICATUS.

BY THOMAS SAVILL, M.D.LOND., D.P.H.CAMB.

MUCH difficulty surrounds the subject of the localisation of the cerebral centre for tactile sensation. In animals it is purely a matter of inference whether the animal has lost the power of feeling when he is touched or pricked, and in man it is necessary that the mental faculties shall be, at least to some extent, retained. Frequently the anæsthesia, if present, is so mixed with other phenomena, and attended by clouding of the intellect, that it is most difficult to come to a conclusion; no apology therefore is needed for placing the following case on record.

Some observers have placed the area for common sensation in the central region of the cortex, that part beneath the parietal bone, corresponding very nearly with the motor area, this, however, is now considered doubtful.

Ferrier was the first and for a long time the only investigator¹ who showed that lesions of the gyrus hippocampi caused anæsthesia of the opposite side.

The laboratory experiments of Horsley and Schafer² localise the centre for painful and tactile sensations also in the gyrus fornicatus, the convolution adjacent to the corpus callosum, on the median aspect of the hemisphere. Thus it would seem that the centre for sensation is located in the whole of the falciform lobe, and not exclusively in either the gyrus hippocampi or gyrus fornicatus.

In the autumn number of *BRAIN*, 1891,³ I have recorded a case containing pathological evidence in support of the

¹ "Functions of the Brain," 1st edition, 1876.

² *Transactions of the Royal Society*, Vol. 179 (1888), B., pp. 1-45.

³ *BRAIN*, Part LV., 1891, p. 270.

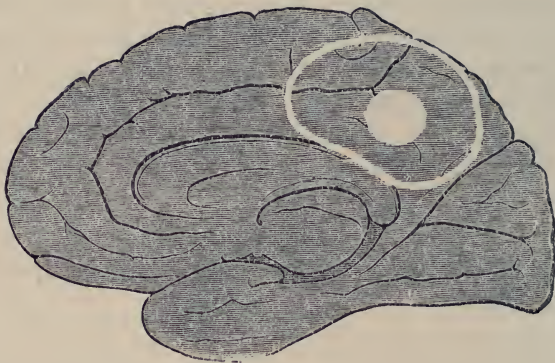
latter experiments, and it is in the belief that the following case adduces evidence of a similar nature that I am about to narrate it.

John W—, aged 47, a hawker, was admitted into the Paddington Infirmary on January 1st, 1892. He had drank a good deal of beer and spirits, but had had no previous illness until three years before admission, when he first began to have fits. He usually had fits in series of ten or more at a time; and, after one of these attacks nine months ago, the left hemiplegia, for which he was admitted, came on quite suddenly.

The patient on admission was quite rational, and when examined by my colleague, Dr. R. D. Hotchkis, was found to have partial loss of power and some rigidity in the left arm and leg, and it was noted that the sensation of the left arm was considerably diminished as compared with the right. Tactile sensation was very nearly but not absolutely lost in the left arm, normal in the left leg and elsewhere. The knee-jerk was normal on the right side, diminished on the left. No ankle clonus. There was weakness, but considerable power remained in the left arm and leg, and he was able to walk without assistance. The trunk seemed very weak, though we could not tell which side was worst, for he had difficulty in sitting up.

The left pupil was larger than the right, but there was no paralysis of cranial nerves. Five days after admission he had a fit. It was heralded, as usual, by a feeling of "cold water being poured on the left big toe, and running up the left side." First the left shoulder began to twitch, then the left leg was drawn up, and at the same time clonic spasms jerked the head and eyes to the right. This was immediately followed by general clonic spasms of the left arm and leg, the right arm and leg being meanwhile in a state of tonic rigidity. The eyes were wide open, conjunctivæ sensitive, teeth grinding, but the patient was completely unconscious. After lasting five minutes he recovered completely. He had another fit on the seventh, two on the eighth, one on the tenth, and one on the twelfth day after admission. He was extremely bad tempered, and became so unmanageable on the 16th January that he had to be transferred to the Workhouse. Three days later he became comatose, and after remaining in a condition of coma for seven days he died, without, so far as we were aware, any modification in his symptoms. The urine and viscera were noted as normal all the while. The temperature while in the Infirmary had a tendency to be subnormal. It was not taken in the Workhouse during the last week of life.

At the autopsy, made forty-eight hours after death, a tumour was found occupying the interior of the posterior segment of the brain, coming to the surface and involving the convolutions, as shown in the accompanying diagram. The other organs were natural, and there were no secondary deposits. The tumour was a round-celled sarcoma, about the size of a small orange, more or less spherical in outline, but its boundaries were not at all clearly defined anteriorly, infiltrating the adjacent tissues, especially at its upper and anterior boundaries. The light spot in the diagram



Median surface of right hemisphere of J. W—, aged 47. The light spot indicates the position where the tumour protruded from the surface. The line around indicates a zone of softening and congestion which projected above the level of the median surface.

indicates the position where the tumour protruded from the surface. The line around indicates a zone of softening and congestion which projected above the level of the parts around. This zone corresponds to the paracentral and quadrate lobules, and the posterior third of the gyrus fornicatus. The tumour also projected into the posterior and superior wall of the lateral ventricle. On the vertex of the brain the tumour did not appear, but the posterior third of the hemisphere was congested.

Remarks.—The diffuse nature of the anterior and upper parts of the tumour prevented one from saying very precisely which of the white fibres of the corona radiata were destroyed or involved; but it was quite certain that the grey matter of the posterior third of the gyrus fornicatus, the

greater part of the quadrate lobe, and the posterior extremity of the marginal convolution, was considerably disorganised and its communication with parts below cut off. Indeed the tumour fungated out on to the median aspect of the first-named convolution, which was the chief one involved. When read in connection with the experiments, and the case alluded to at the outset, one is justified in believing it *probable that the posterior part of the gyrus fornicatus is the cerebral centre for the tactile sensation of the arm.*

The paralysis from which the patient suffered was only very slight, and was quite in keeping with the supposition that the white motor strands were pushed aside, rather than destroyed, by the infiltrating growth.

As regards the "fits," which I had the opportunity of observing myself, it would have been somewhat difficult, from their character, to have localised the seat of the tumour. But this uncertainty is accounted for, as above mentioned, by the diffuse character of the anterior extremity of the tumour.

CASE OF HOMONYMOUS HEMIANOPSIA, WITH POST-MORTEM APPEARANCES.

BY EDWIN L. DUNN, B.A., M.B., ETC.

Assistant Medical Officer, Wakefield Asylum.

M. A. S., æt. 43, was admitted to the Wakefield Asylum on December 15th, 1891.

History.—Patient has been in the habit of giving way to bouts of alcoholic excess. For about a month before admission she had been drinking very heavily. About one week before admission she was noticed to be “paralyzed on the left side, the hand was drawn up and the leg useless.” Nothing of her family history known.

On admission she was in a maniacal condition and raving wildly. No direct reply to queries could be obtained. It was noticed that there was paresis of the left side, with slight rigidity of elbow and shoulder joint. The grasp of left hand was very weak in comparison with the right. She could walk only with assistance, and the left leg was raised with difficulty and set down with a jerk on the heel. There was constant convulsive twitching of left leg and arm. The pupils were equal and semi-dilated. Their reactions were normal.

December 28th, 1891.—It is noticed that there is anæsthesia (to prick of a pin) of the left side, strongly marked in arm and leg, but not so much in face. There is possible slight ptosis of the left eyelid.

January 3rd, 1892.—Pupils are equal, all reactions present. The tongue is protruded to the left side. Sensation in left arm and leg is much better, being almost normal. Plantar reflex not well marked on left side; more marked on right, where it is, perhaps, excessive. Knee-jerk present on both sides, more marked on left. No clonus either side.

10th January, 1892.—Patient is much calmer. There is no defect of hearing. Sensation to pin-prick normal. Plantar reflex is excessive on both sides. There is no clonus. Knee-jerks normal. Rigidity of left arm almost disappeared. Grasp of left hand is weaker than right. There is no rigidity of left leg.

The gait is steady, but the left leg is moved in rather a jerky fashion, and there is a tendency to come down on the heel. There is no facial palsy. The tongue is protruded quite straight.

Eyes.—There is left homonymous hemianopsia, the defect being limited to the lateral half fields of vision in each eye. There is no change in the hemianopsic field with colours red or green. There is no colour blindness. Wernicke's pupillary inaction sign is not present (the pupil contracts when light is thrown on the blind half of the retina). Pupillary accommodative reflex present. Light reflex present, but sluggish. Concentrated light also sluggish. Consensuals present, but very sluggish in left eye. Pupils do not dilate to skin stimulus; they dilate at once emotionally. All ocular movements are perfect.

January 15th, 1892.—Hemianopsia unchanged. She says her leg is "all right," but complains that the left arm is stiff and the hand occasionally numb. She is fairly rational, but prone to emotionalism.

January 24th, 1892.—It is noticed that she is prone to drop pieces of bread, plates, &c., out of her left hand. She complains that it does not feel right. Grasp on both sides almost equal. She constantly leaves food which has dropped to the left side of her plate.

February 13th, 1892.—Mental condition and hemianopsia as before.

February 20th, 1892.—The left arm has never completely recovered its normal amount of motor power, but has always been slightly weaker than the right. Sensation to painful stimulus has remained unimpaired. She now complains that the arm is very numb, and is getting weaker. She has intense right-sided headache.

February 25th, 1892.—The headache has continued, the arm has become much weaker. Both pupils are widely dilated, light and consensual reflexes are present.

February 26th, 1892.—Patient became suddenly maniacal last night. To-day she cannot respond to queries, and is raving continually.

March 4th, 1892.—Mental state as above. The left arm and leg have been gradually getting worse, and are now completely paralyzed. Knee-jerk is present in both legs, is perhaps slightly increased in left. Plantar reflex is absent in left, present in right limb. There is some rigidity in paralyzed limbs, especially in the arm. Sensation cannot be tested owing to the mental state.

March 16th, 1892.—Sensation to prick of a pin is fairly per-

fect. She cannot tell what it is; says it is a "razor." She can just move left hand and can draw up the left foot one inch.

March 20th, 1892.—She had a slight stroke or syncopal fit yesterday. (Pallor, lividity, coldness of extremities, &c.) The left pupil is now widely dilated. Reacts slowly to concentrated light. The right pupil is semi-dilated, reacts quickly to concentrated light. Both appear to accommodate slightly and sluggishly. The optic discs and retinae are found normal by ophthalmoscopic examination. The paralysis in arm and leg is incomplete as before. Sensation to painful stimulus appears perfect.

April 2nd, 1892.—Died of pneumonia, her condition not having otherwise changed.

Autopsy.—Cardiac valves and muscle healthy. Right lung congested with small spot of grey hepatization in middle of inferior lobe. Left lung solid and in a condition of grey hepatization. Liver and kidneys shew fatty change. Spleen soft and friable.

Skull cap thickened uniformly. Dura mater normal. No adhesions anywhere. Lepto-meninges thickened and softened, from right hemisphere peeled with undue ease, except from parietal and occipital lobes.

Right hemisphere of cerebrum.—The part of this hemisphere, from and including ascending parietal gyrus backwards, as compared with the left is notably swollen, softened, and collapsed. The pia cannot be stripped therefrom. Of this the superior parietal lobule, quadrate, first annectant gyrus, and lower portion of angular gyrus bounding the parallel sulcus, are the most disintegrated. The superior parietal lobule in front of the parieto-occipital fissure, shews a spot of ochre-coloured softening about the size of a threepenny-piece.

The supra-marginal gyrus consists of two parallel gyri taking origin from ascending parietal; the superior of these two is more softened than the inferior.

In the occipital lobe the superior occipital convolution is the most softened, and the mesial surface is not so softened as the outer. The ascending parietal is softened in its upper two-thirds only (*i.e.*, the part above the origin of the supra-marginal gyrus). The whole convolution is atrophied. The posterior third of the gyrus fornicatus is softened. The superior temporal gyrus is softened; its posterior two-thirds especially. The remaining temporal gyri and the frontal lobe appear healthy.

On horizontal section of brain the centrum ovale majus shews

softening in its posterior half. On further section the softening is found to involve the white matter of the parietal lobe and of the anterior two-thirds of the occipital lobe. The posterior third of the latter, though soft in comparison with the opposite side, is distinctly less affected than the parts first mentioned.

A section through the hemisphere passing through the three portions of the lenticular nucleus at the level of their greatest transverse diameters, shews that the greatest softening involved the posterior third of the roof of the descending horn, and the entire roof of the posterior horn of the lateral ventricle (*i.e.*, tapetum, optic radiations, and rectangular bundle of Wernicke). The white matter of posterior portion of occipital lobe is still of fair consistence.

On deeper section the tapetum, optic radiations, and rectangular bundle are still more disintegrated, readily breaking down under water; there is a small cyst the size of a pea in the posterior part of the roof of the descending horn near the margin.

Left hemisphere.—The convolutions are rather small, otherwise there is nothing abnormal.

No ruptured or thrombosed vessels discovered. Basal vessels healthy.

The cerebellum, pons, medulla and spinal cord shew no naked eye evidence of disorder.

Remarks.—It is possible that the primary lesion in this case was at the seat of the cyst in the posterior part of the roof of the lateral ventricle. We may regard this as the cause of the hemianopsia which was recognized early in the case and remained unchanged throughout. The cortex of the occipital lobe and the white matter immediately underlying the same—though undoubtedly affected by softening, was probably not so to such a degree as to cause well-marked symptoms. It is a noteworthy point that, although the angular gyrus was affected to an extreme degree, there was nevertheless no crossed amblyopia. Such a condition might possibly have been present on admission, but certainly was not so as soon as the patient's mental state permitted thorough examination.

With the condition of the left pupil, which was widely dilated for a fortnight before death, it is interesting to compare a case of Dr. Delépine's (read before the Pathological

Society of London, May 20th, 1890), in which there was right homonymous hemianopsia with dilatation of the right pupil—the *post-mortem* appearances being almost complete destruction of the left cuneus by softening, and also softening in the middle part of inferior frontal and posterior orbital convolutions, and another softening at the bottom of the fissure of Rolando, opposite the posterior end of superior frontal sulcus.

I regret exceedingly that sensation was not more thoroughly tested. The patient's mental state, however, and her extreme emotionalism rendered any lengthened examination requiring her attention extremely difficult. Sensation to the prick of a pin was normally present throughout except for the first few days. This fact, as far as it goes, is opposed to the views of Munk, Luciani and others, who hold that the localization area for common sensations lies in the central region of the cortex just beneath the parietal bone, a region in this patient extensively diseased. It is interesting, however, in connection with the experiments of Horsley and Schäfer (*Trans. Royal Soc.*, vol. 179 (1888), B., pp. 1-45) who hold that the centre for painful and tactile sensations is in the gyrus fornicatus. Though part of that convolution was in this case diseased, there is no reason to suppose that sufficient healthy tissue was not present to functionate correctly.

As to the awkwardness and subjective sensations of the left hand, this is an interesting point, and was a prominent feature of the case throughout that period in which the patient was fairly rational. She constantly complained to the nurse that she had not proper use of her left hand, that it was numb and not like the other—this being a source of great distress to her. Her awkwardness too in holding her sewing, and her tendency to let objects drop was remarkable, although at this time the grasp of the hand was fairly good and there was little diminution of the motor power of the arm. In this connection it is interesting to note that Bernhardt (*Archiv. fur Psychiatrie*, vol. xii., p. 780) reports four cases in which there were subjective affections of sensibility

FIG. 1.

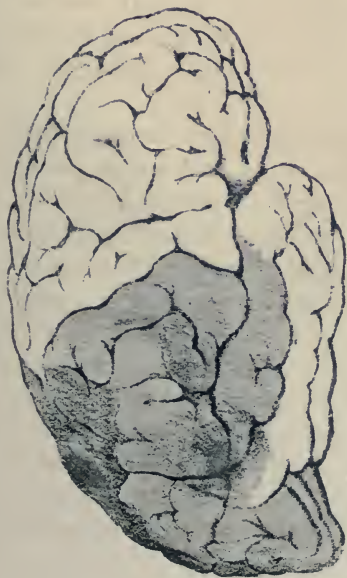


FIG. 2.

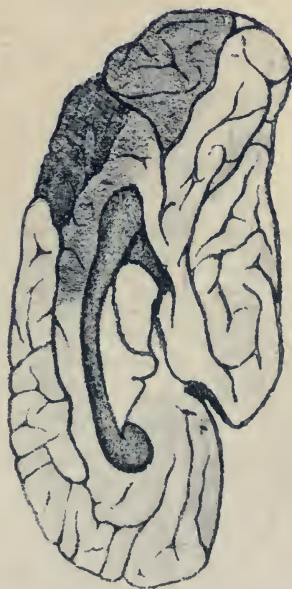


FIG. 1.—Exterior surface of R. hemisphere.
FIG. 2.—Mesial surface of R. hemisphere.

FIG. 3.



FIG. 3.—Deep horizontal section of R. hemisphere, shewing softening in posterior part of roof of Lat. Vent.

Depth of shading indicates degree of softening.

and impairment of the muscular sense associated with hemianopsia, and compares these with a case of Samelsohn's, shewing similar disturbances. He considers that these cases belong to a type in which after an apoplectic attack the symptoms of paralysis disappear, leaving a characteristic awkwardness in the limbs of one side, especially the upper extremity. This awkwardness is further aggravated by the hemianopsia, a frequent symptom in these cases. These cases of Bernhardt's have not as yet been completed by *post-mortem* examination. It is worth while also to compare this case with one of Westphal's (*Charité Annalen*, 1882), in which there was marked impairment of the muscular sense of the right upper extremity, associated with diminished right-sided sensibility and bilateral right hemianopsia. The lesions found after death were softening and atrophy of the whole parietal lobe (including ascending parietal), the greater portion of the occipital and a small portion of the temporal lobe, the cortex only being involved.

The phenomena exhibited by the left plantar reflex (which was at first deficient, then present, and disappeared totally after the second maniacal attack, about one month before death), may be of interest in connection with the disease of the white and grey matter of the opposite hemisphere and the macroscopically healthy basal ganglia.¹

¹ Königsdorf (*Deutsche Med. Wochenschrift*, March 3rd, 1892) reports a case of hæmorrhagic encephalitis in which, with much softening and blood extravasation into the grey matter and white substance of the left hemisphere, the basal ganglia also being affected, there was three days before death complete absence of all superficial reflexes on the right side.

A CASE OF CEREBELLAR TUMOUR, WITH LOSS OF THE KNEE-JERKS.

BY HENRY HANDFORD, M.D., M.R.C.P.LOND.

Physician to the General Hospital, Nottingham.

CASES of brain tumor, are always interesting, on account of the wonderful variety of symptoms to which they give rise, dependent not only on the position of the tumour but also very largely on its degree of vascularity, its rapidity or slowness of growth, and on pressure, sometimes affecting parts at a considerable distance, sometimes in an inexplicable manner, doing but little damage to nerves or tissues in close proximity to or involved in the growth. And, further, the symptoms are apt to be complicated by secondary meningitis which so frequently arises, and by the accumulation of fluid in the ventricles, or in the sub-arachnoid spaces. But however interesting the individual problems may be, it is not on that account that I bring forward the following case, but rather as a contribution to the elucidation of the vexed question of the physiology of the knee-jerks.

Case.—L. W., aged 16, was admitted on March 2nd, 1891. His illness began three months previously, with sickness coming on many times daily, independently of taking food. This was followed by headache, chiefly frontal, giddiness, unsteadiness of gait, and occasional tingling in the hands and feet. For the ten days before admission he suffered from fits, commencing with pain in the back and head. He then suddenly “shoots himself out straight” and lies thus quite still with his eyes wide open for ten to twenty minutes. Beyond very slight twitchings of the hands no spasmodic movements or muscular contractions take place during the fits, the limbs remaining flaccid. For the last few months his sight has been failing, and for the past four or five days he has seemed “strange in his mind.” He is very slow in answering a simple question. On admission it was noted that the knee-jerks were completely absent on both sides. They

were thoroughly tested, as I was much surprised at the fact, and could not then offer a satisfactory explanation. The plantar and abdominal reflexes were present on both sides, and there was very slight ankle clonus also on both sides. There was no anæsthesia or analgesia. The other systems were normal, and the urine was free from albumen and sugar. Double optic neuritis was found, more advanced in the right eye, and by March 9th some weakness of the ocular muscles, especially of the abductors, and slight nystagmus were noted. The pupils were dilated and reacted imperfectly to light. There were numerous small hæmorrhages in the retinæ. He could still see a little, but eventually he became quite blind. He was rather feeble, but there was at this time no definite local paralysis. His gait shewed much inco-ordination and unsteadiness with a tendency to stamp the heels. He had great difficulty in turning round, and complained much of giddiness. He could stand with difficulty with the feet apart and the eyes closed, but with the feet close together he tended constantly to fall *backwards*. He had never fallen whilst walking with the eyes open. On March 13th there was marked internal strabismus of the right eye, but it was temporary.

April 4th.—The patient gets rapidly worse. He screams a great deal, and lies in a semi-conscious state. There is a good deal of retraction of the head. He has had several more fits. Knee-jerks still quite absent, though the plantar and abdominal reflexes are still brisk. No ankle clonus can now be detected on either side. On account of the imperfect consciousness it is very difficult now to determine the condition of sensation. He cannot move body or limbs. He is emaciating rapidly. The temperature has all along been normal.

After this the paralysis became more and more absolute, difficulty in swallowing arose, and also attacks of dyspnœa, and hurried respiration, apparently from implication of the respiratory centre. Although emaciation was extreme, and he had been accustomed to pass urine and fæces into the bed for several weeks, no bed sores formed. The limbs remained flaccid. He died on May 31st. Total duration of symptoms six months.

Unfortunately the *post-mortem* examination took place when I was out of town for a day or two, and so the spinal cord was not examined.

There was no meningitis, the cerebral convolutions were somewhat flattened, the lateral ventricles distended with fluid, and a tumour was found about the size of a large walnut on the under

surface of the cerebellum growing from the middle lobe (fig. 1). It was pressing on the medulla, which was somewhat flattened, and it had closed the foramen of Majendie, thus causing the distension of the lateral ventricles. It did not involve any of the cranial nerves (fig. 2). On antero-posterior vertical section the tumour was found to be more or less defined, though not encapsuled. It had destroyed the whole of the middle lobe of the cerebellum and extended into both lateral lobes, especially the left. On microscopic examination it proved to be a sarcoma. There were no other growths in any of the other viscera.

The physiology of the knee-jerks is still obscure. It is agreed that the integrity of the reflex arc, consisting of afferent nerves, ganglion cells in the anterior cornua, and efferent nerves is necessary, though it is denied that the movement is a true reflex action, because the time elapsing between the stimulus—the blow on the tendon—and the jerk of the leg is much too short. But it is not agreed whether anything further is necessary for the normal activity of the knee-jerks, nor why they become excessive in some cases and fail in others, the reflex arc remaining uninjured.

According to one school the spinal reflex arc is sufficient by itself for the production of the knee-jerks, and it is habitually controlled by higher encephalic centres. When this controlling influence is cut off by a cerebral lesion, or by a total transverse lesion of the cord, exaggerated reflexes result.

On the contrary, the French school teach (and their views have been largely adopted in this country), that the degenerative changes in the terminal portion of the fibres of the crossed pyramidal tracts “cause an irritative overaction in the related great ganglion-cells in the anterior cornua, and thus lead to an exaggerated condition of ‘tonus’ in the muscles and the increased tendon reactions.”

Dr. Hughlings Jackson has taught for many years that muscular “tone” and the tendon reactions depend on cerebellar influence, which is normally more or less antagonised by the influence of the cerebrum. And when the influence of the cerebrum is cut off by any lesion “the unantagonised influx of cerebellar energy” is the cause of the increased “tonus,” tendon reactions, and rigidities.

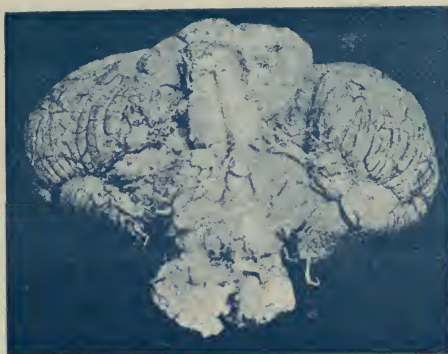


FIG. 1.

Photograph of the under surface of the Medulla, Pons, and Crura lying upon the Cerebellum, the middle lobe of which is occupied by a tumour projecting chiefly to the right. (*Hold at two feet distance.*)



FIG. 2.

Vertical antero-posterior mesial section shewing the extent of the tumour and the flattening of the Medulla. The tumour is dark.

Dr. Bastian¹ has adopted a somewhat similar view, and has much strengthened his position by showing that in "total transverse lesions of the spinal cord," when the influence both of the cerebrum and of the *cerebellum* is entirely cut off—the knee-jerks and all reflex actions concerned with parts of the cord below the lesion are completely absent, notwithstanding the most marked descending degeneration of the crossed pyramidal tracts.

I have quite recently seen a case of fracture of the sixth cervical vertebra, causing a total transverse lesion of the cord. There was complete paralysis of motion and sensation of all kinds up to the level of the nipples. Twenty-four hours later the paralysis had involved the arms. The man died in forty-eight hours, but although all appearance of "shock" was absent after the first few hours the knee-jerks were absolutely gone.

The case of cerebellar tumour that I have described seems to have almost the value of an experiment in shewing the connection of the cerebellum and the knee-jerks. According to widely accepted views the pressure of the tumour on the medulla should have produced descending sclerosis of the crossed pyramidal tracts and *exaggerated knee-jerks*. The presence of optic neuritis gave rise to the suspicion of a tumour, and the inco-ordination of gait and tendency to fall *backwards*, suggested the middle lobe of the cerebellum as the seat. But the complete absence of the knee-jerks in that early stage when the lad was still able to walk was a great difficulty in accepting the diagnosis of brain tumour. The inco-ordination and the stamping of the heels much resembled the gait in locomotor ataxia, a disease with which, according to some, the cerebellum has much to do.

At this time there was complete control over the sphincters; there were no shooting pains, no loss of light reflex, no muscular wasting, and no loss of sensation; and the absence of bed sores, even to the end, showed that trophic impairment was not great. Under these circumstances it is scarcely possible to suppose that there could

¹ *Medico-Chirurgical Transactions*, vol. 73, p. 207, *et seq.*

have been any peripheral neuritis, sclerosis of the posterior columns of the cord, degeneration of the posterior nerve roots, or any tumour or other coarse lesion of the cord or cauda equina to account for the loss of the knee-jerks. And, besides, with the exception of this one disputed question of the knee-jerks, the cerebellar tumour and the consequent distension of the lateral ventricles, were quite sufficient to account for all the symptoms from which he suffered. There seems, therefore, much reason to believe that the loss of the knee-jerks in this instance was due to the cutting off of the cerebellar influence, by the growth of the tumour.

LARGE TUMOUR OF THE PITUITARY BODY, INCREASED KNEE JERKS, NO ACROMEGALY, NO GLYCOSURIA.

BY HENRY HANDFORD, M.D., M.R.C.P.LOND.

Physician to the General Hospital, Nottingham.

Case.—E. S., aged 53, single, was admitted in July, 1891. She began to lose the sight of the right eye last Christmas. The left failed at Easter, since which time she has been unable to follow her employment as a lace worker. Vomiting has been frequent, and she has suffered greatly from headache, chiefly affecting the right parietal region, which is tender to the touch. She lies in a drowsy state with the eyes closed. Memory is very defective, but otherwise her intelligence is acute, and it remained so until the end. There was no trace of acromegaly; and the urine remained free from sugar as well as from albumin. The eyelids could be raised but not quite fully, and there appeared to be some weakness, though the patient professed that she kept them closed voluntarily. There was also slight weakness of the internal recti, and some nystagmus on looking towards the right, none on looking towards the left. The pupils were equal, one half dilated and insensitive to light. The patient was blind but could distinguish day from night, though she could not distinguish the position of the windows. There was optic atrophy, with white discs, rather small vessels, but no trace of previous neuritis. At this time there was some doubtful weakness of the left side of the face, which a few weeks later became marked. There was no definite weakness of the limbs, the patient being able to walk fairly and use both hands. There was no alteration of sensation. The plantar reflexes were not noticeably increased, but the knee jerks and arm jerks were exaggerated. The bowels were exceedingly obstinate, being only moved after croton oil. She had no fits, but frequently had attacks of stertorous breathing which lasted several hours.

On August 31st, it was noted that the tongue deviated towards the right, and that the right half appeared wasted. She was much more feeble and could hardly stand. By September 21st the left arm and leg appeared decidedly weaker than the right, but on October 1st this difference could not be definitely estab-

lished, so much did the degree of paralysis vary. Taste was impaired and she called salt sour; but other things she recognised. Hearing remained acute. Speech became more and more thick and eventually she lapsed into a comatose state and died on October 17th, three and a half months after admission, and ten months after the first onset of symptoms.

At the necropsy a large tumour was found at the base of the brain pressing on and causing considerable flattening of the Pons Varolii, especially in its superior half. On antero-posterior vertical section the tumour was found to have originated in the pituitary body, and to be more or less encapsuled. The arteries of the circle of Willis were imbedded in the middle of the growth. The optic nerves and commissure were pressed very flat, and were partially involved in the tumour. The 3rd and 4th nerves were flattened and wasted, and the crura cerebri were also flattened and much atrophied. The tumour was a sarcoma. There were no growths in any other parts of the body.

The only mental symptoms here were some oddness of manner, great loss of memory and hebetude. It is remarkable that so large a tumour in that position should have caused so little injury to the cranial nerves. The optic atrophy was probably due to the pressure on and wasting of the optic nerves and commissure, and was not the result of previous papillitis, because the vessels were very little diminished in size, and there was no trace of lymph remaining. With this exception none of the other cranial nerves were *completely* paralysed. So far as the rest were affected the paralysis was *one sided*, or *variable* and incomplete, although the tumour was large, centrally placed, and nearly symmetrical. Although the crura cerebri were so much flattened there was little paralysis of the limbs until the last week or two, and no rigidity. The knee jerks and the reflexes of the upper extremities were increased on both sides as would be expected. It is to be noted that while the cerebral influences were largely cut off by the pressure on the crura the cerebellum and its connections with the cord were not affected.

The associations of tumours of the pituitary body or hypophysis cerebri, both with acromegaly and with glycosuria make the absence of both these conditions in this case worthy of note.



FIG. 1.

Photograph of the base of the brain. The whole of the middle fossa is occupied by a mushroom-shaped tumour. (*Best seen at two feet distance.*)

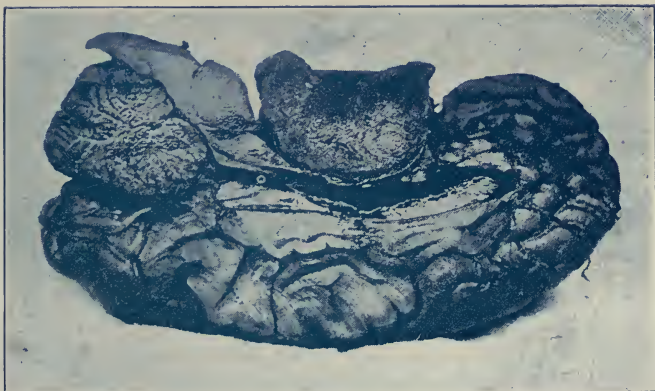
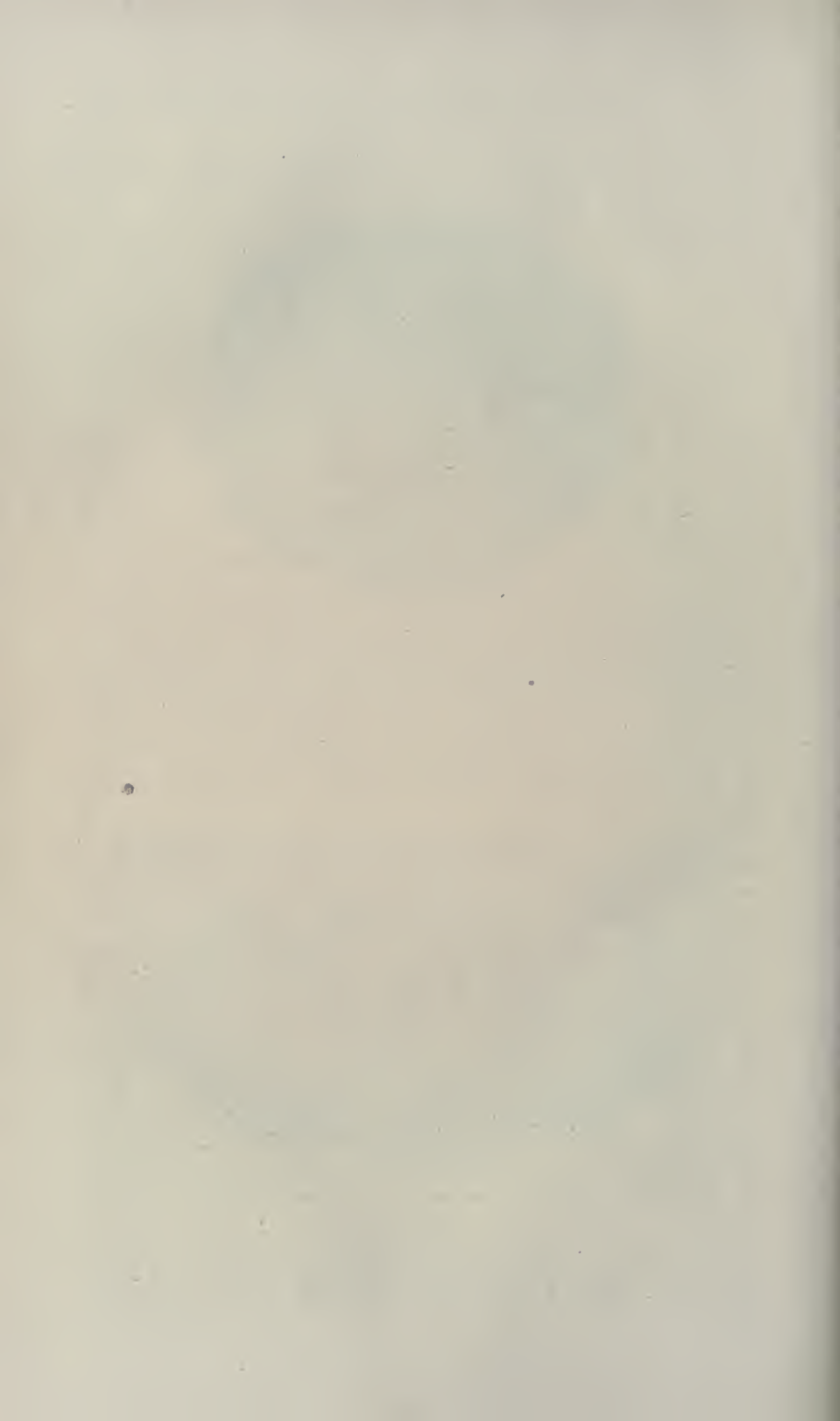


FIG. 2.

Vertical antero-posterior mesial section shewing the flattening of the Pons Varolii and of the Crura Cerebri by the pressure of the tumour which is growing from the Pituitary Body.



TUMOUR OF PITUITARY BODY WITHOUT ACROMEGALE.

BY ERNEST WILLS, M.D.LOND.

Assistant Medical Officer, County Asylum, Rainhill.

J. G., æt. 26, cotton operative, was admitted into Rainhill Asylum, April 20th, 1891, suffering from dementia; he was very confused, his memory was defective, rendering him incapable of giving any good account of himself, but he complained much of pains in his head. His facial expression was dull, somewhat anxious, and his general appearance cachectic. He looked younger than his age, was 4ft. 10in. in height and weighed 100lbs. His brother, who accompanied him to the asylum, also a short man, but taller than patient, healthy and fairly intelligent, gave the following information :

Family History.—They were both illegitimate children, mother dead, cause unknown, father living, healthy, now married. No insanity, consumption or cancer in family on either side as far as informant knew.

Previous History.—Patient had never had any severe illnesses, or chest trouble. He had complained of pains in his head since he first began work at the mills fifteen years before. During the last six months they had become much worse, causing him to cry out at times “Oh, my head,” and he had become very stupid. There had been no previous attack of insanity and no fits.

Condition on Admission.—Temperature normal. Well nourished generally. Skin elastic, subcutaneous fat abundant. Circulatory system, heart sounds normal, pulse regular, moderate tension 68. Respiratory system normal. Abdomen normal. No signs of syphilis. No albumen or sugar in the urine. Nervous system. External muscles of right eye normal, some paresis of left internal rectus with external strabismus. Irides hazel, margins circular and regular. Right pupil reacts to light and accommodation,

left scarcely to either. Very little if any vision with the left eye. No paralysis of facial muscles, tongue protruded in middle line. No marked paralysis of limbs, but the grasp is distinctly feeble on both sides and a little weaker on the left than right. He is feeble on his legs, walking with a staggering gait and slight tendency to fall backward. Reflexes: the knee-jerks are present on both sides, the left being somewhat excessive. No ankle clonus. Superficial reflexes present, average, no marked difference between the two sides. Sensation to touch pain, and temperature is good all over. No hyperæsthesia. There is consecutive optic atrophy of considerable duration in both discs, but especially well marked in the left.

April 30th.—Very dull and heavy, lies in bed taking no notice of surroundings, crying out with pain occasionally. Has vomited two or three times. Has had no fits.

May 5th.—Brighter and more lively; although still confused and complaining of headaches, says the pains are less severe.

May 12th.—Complains of recurrence of severe pains in the head and also in region of the left elbow; there is no neuritis. Vomited once to-day. No fits.

May 16th.—He was very dull and lethargic, and had retraction of the head. After some hours he became comatose and died at night somewhat suddenly.

The following account of the tumour is from the report of the autopsy Dr. Gordon Sanders' (late pathologist to the asylum).

Post-mortem Examination.—Body squarely built, the hands short, broad and somewhat spade like, but all limbs fairly proportioned. There was a very large subcutaneous deposit of fat. There was nothing of any importance in any of the organs except the brain. The scalp was thick and hairy, the skull low, broad, rounded, of good symmetry, and natural thickness. On opening the skull and dura mater the cerebral hemispheres were seen to be flatter than natural; on attempting to remove the brain a tumour was found at its base, in the region of the infundibulum, consisting of a dark soft greenish-yellow mass. The tumour could not be removed entire as a large part of it was wedged in the sella turcica, it was therefore cut across and this part dissected out afterwards. The brain itself seemed fairly normal, and presented no irregularity of convolutions, although on the vertex they seemed somewhat swollen and flat with considerable distension of veins; there was distinct diffused opacity of membranes, however, with much adhesion, in many parts reaching actual decortication, especially at the vertex. At the base of the brain within the

interpeduncular space lay the tumour, extending anteriorly as far as the posterior termination of the orbital surface of the frontal lobes. Laterally it was overhung by the temporo-sphenoidal lobes. Posteriorly it extended to and separated widely the crura cerebri. The greater mass of its substance lay eccentrically to the left and on separating the (apparent under but real) upper surface was found to lie in a hollow between the left frontal lobe and the left crus cerebri.

Position of Structures Round.—Anteriorly the two *olfactory nerves* were thin and flattened, the left much the more so, of which the external root was alone visible as a thin whitish band. The right *optic tract* skirted the right side of the tumour and was involved in the thickening of the capsule, the half of the optic chiasma on that side was seen lying just anterior to the projecting part of the tumour; it was white, tough and atrophied. The *left optic tract* was very remarkably altered. It passed over the crus as a pale yellowish thin band and at once became involved in the tumour mass, being distinguished as a soft yellowish ill-defined band passing forward obliquely to join the right tract in the chiasma, the left half of the chiasma being soft, yellowish-grey, and translucent in marked contrast to the right. The *crura cerebri* were widely separated, the right was flattened and wasted, the left more displaced outwardly, more rounded and prominent. The *corpora albicantia* were pushed to the right side and much flattened, the right being nearly half the size of the left. The floor of the third ventricle and interpeduncular space seemed softened by pressure, but were not implicated in the tumour. The *pons* seemed wasted and narrowed, the ventral pyramids were prominent, but the mesial sulcus was deep and the sides flattened. The *medulla* was possibly somewhat atrophied.

Description of Tumour.—It consisted of two parts connected by a pedicle, the upper part was removed with the brain, the remainder lay in the sella turcica and was dissected out after, and the sphenoid bone removed. The portion of tumour removed with the brain consisted of a soft glistening body the size of a Tangerine orange irregularly rounded or ovoid, about 3.5 cm. in diameter and was divided into two unequal parts; a soft flatter darker basal portion of diameter referred to, and a more prominent hard yellowish spongiform body the size of a marble, somewhat gritty, seated in the right anterior segment (apparent left, as the brain lay with tumour uppermost), separated by narrow neck from the basal portion. This second projecting portion was that which was continuous with the pituitary body and infundibulum. The

whole tumour had a thin distinct capsule unequal in thickness, more ill-defined and grayish over the basal part, but tough, thicker and whitish over the dependent portion, from which it was easily separable. The flatter basal portion was of varied colour; the left half being of greenish yellow hue, somewhat like a greengage in colour, across it posteriorly lay a large whitish yellow band, which proved to be the left optic tract; the right half was more distinct, yellower and firmer.

The bony floor of the sella turcica presented sharp elevations and ridges, and when the dura mater was stripped from them their summits were found rough and granular. The sella turcica was much broadened and its constituent bones remarkably thin, the basilar portion being a mere lamina and very brittle. The tumour here was very soft in its anterior half and lay entirely enclosed in a thick capsule, the posterior half was of deep canary yellow, rough, spongioid and the seat of a fairly recent hæmorrhage. The anterior half or section was found to be a cyst containing a greenish yellow glistening fluid somewhat viscous. Although the floor of the sella turcica was pitted in several places into cavities the size of a split pea, at no point was there any communication with the buccal cavity from which the tumour was shut off by a mere shell of bone.

Microscopic examination of various parts of the tumour showed that it was composed of two parts, a minor solid and a much larger part, in fact the majority of the tumour, which was cystic. A section of the solid or semi-solid portion of the tumour exhibited a structure resembling that of the thyroid body, viz., a capsule of fibrous tissue with cleft like spaces in it lined with flattened epithelium, an inner stroma of fibrous tissue containing small cysts of various sizes lined with columnar epithelium and the lumen of the cyst containing a viscid substance or filled with cells columnar, ovoid, or round. Nowhere in the specimens were any columns of cells spreading in a malignant manner, although there were here and there to be seen groups of cells somewhat resembling the nests in a carcinoma, these were probably only the walls of smaller cysts in the plane of the section.

Sections of the walls of the larger cysts only showed a fibrous structure, with the cleft like spaces as mentioned above, and with patches of pigment here and there, the remains probably of old hæmorrhages as the fibrous tissue was very vascular.

Commentary.—This case has several points of great interest which make it worthy of record. 1. Clinically from the large size it attained, and the amount of displacement of surrounding parts it caused without giving rise to any serious symptoms until quite late in the case. 2. The nature of the tumour itself. 3. The absence of any acromegale in association with the tumour of pituitary body.

Clinically the principal symptoms were those usually found with cerebral tumour, headache, vomiting and optic neuritis with consecutive atrophy; but until his mental condition caused him to leave off work, he had managed very well as an operative in a mill and at no time had he any paralysis, nor did the almost total loss of sight with the left eye seem to have troubled him.

The organic dementia caused by the tumour is interesting, too, as most authorities agree that cerebral tumour as a cause of insanity is decidedly rare, although insanity as a sequence of tumour is fairly common. With respect to this point it may be mentioned that during the last twelve months there have been an exceptional number at Rainhill Asylum, viz., five cases in about 240 *post-mortems*—this of course is very much above the average.

With regard to the tumour itself, it seems to be what is called by some authors, *gôitre* of the pituitary body from its resemblance to the structure of the thyroid gland. Weigert calls it adenoma of the pituitary body and supposes it to be a cystic degeneration and hyperplastic overgrowth of the anterior lobe, which lobe in the adult normally contains a large number of convoluted tubules or alveoli lined with columnar epithelium which in some cases fills up the tube; moreover portions of the tubules are frequently cut off by the connective tissue so as to form vesicles. The lymphatics of the organ originate in cleft like spaces between the tubules and pass to a network in the capsule (Quain's Anatomy, vol. 2). All these characters are represented in the present tumour. With regard to acromegale, in many cases of that disease which have been published, hypertrophy of the pituitary body has been observed, and Dr. Pierre

Marie, in his paper on the subject, BRAIN, July, 1889, says, "Finally amongst the lesions affecting other organs, and which, after what has been observed in other autopsies, seem to me to be constant in acromegale, must be mentioned hypertrophy of the pituitary body with enormous dilatation of the sella turcica, persistence of the thymus and finally hypertrophy of the cord and ganglia of the sympathetic system." The two latter points were not specially observed, but there were certainly no signs of acromegale about the patient, therefore as a negative case it seemed worth recording.

THE STUDY OF LARYNGEAL PARALYSIS SINCE THE INTRODUCTION OF THE LARYNGOSCOPE.

BY FELIX SEMON, M.D., F.R.C.P.

THE following remarks contain the substance of an article which I contributed to the "Festschrift," dedicated to Professor Virchow on the occasion of his 70th birthday. As this paper in all probability will be accessible to comparatively few members of the profession in this country, a translation of its contents may not be unwelcome to those neurologists who have no time to study the history of this subject in detail. This English edition is on the whole a faithful reproduction of the German original; the introduction, however, which referred more specially to the particular occasion of its origin, has been omitted; and the papers of Wagner, Burger, Krause and Risien Russell, recently published in full, are considered at greater length.

I do not aim here at a complete bibliographical survey of the subject. The period I cover extends from the introduction of the laryngoscope, thirty years ago, down to the end of 1890, in addition to which the four important papers just mentioned and published in the course of 1890 and 1891 are discussed, and my object is rather: to retrace in broad lines the evolution of our knowledge concerning laryngeal paralyses, and to refer in detail to such contributions only which in my opinion have actually contributed towards this development.

Opinions will, of course, differ as to the relative value of the individual contributions and attribute to some a greater or a lesser importance than has been accorded to them in the following lines.

But, as I have contended already on a previous occasion (1), these are points on which everybody has the right to form an opinion of his own; and I can give the assurance that I have followed the best of my judgment, being inspired only by the desire to do justice to everyone concerned.

With the only exception of the subject of benign neoplasms, probably no part of laryngeal pathology has profited so much by the introduction of the laryngoscope, as that dealing with the

motor neuroses of this organ. The course of the other most prominent laryngeal affections, such as tuberculosis, syphilis, malignant neoplasms, &c., always offered points for the diagnosis of the true nature of the local disease. But the main, or rather the only, phenomena by which motor laryngeal paralyses are usually manifested, viz., disturbances of phonation, aphonia, dyspnoea, are of such multifarious significance as to point to an inflammatory lesion or to a new growth, as much as to a paralysis. In pre-laryngoscopic times one had therefore to depend entirely upon mere suppositions concerning the nature of the laryngeal affection met with in such cases, and von Ziemssen is perfectly justified in stating (2) in the introduction to the chapter on Laryngeal Neuroses of his "Cyclopædia," that the older literature on this subject is devoid of any importance.

The distinction of having diagnosed the first laryngeal paralysis by means of the laryngoscope belongs, so far as I know, to my illustrious teacher, L. Traube.

In Götschen's "Deutsche Klinik" (3) he described the laryngoscopic appearances in two cases of aneurism of the aorta, viz., paralysis of the left vocal cord, and in his commentary explicitly stated that these laryngoscopic appearances had at once drawn his attention to the possible presence of an aneurism. Thus already in the first two cases of laryngeal paralysis, ascertained by means of the laryngoscope, the *diagnostic* value of the method was emphasised.

Within the next few years the number of communications referring to motor laryngeal disturbances quickly increased. In 1863, Gerhardt's classic "Studies and Observations of Paralysis of the Vocal Cords" (4) were published, which contain numerous valuable cases and in which the principle already is stated, that in cases of paralysis of the trunks of the vagus or recurrent laryngeal nerves, individual strands of fibres may be affected singly or at least predominantly, a statement which was opposed very energetically six years later by Navratil, (5) and as late as seventeen years afterwards by Bosworth. (6)

During the years 1863 to 1876, a large series of valuable communications were published, which increased our knowledge concerning the individual forms of laryngeal paralysis, without however, formulating any general principles concerning the laws governing the production of these different forms. Apart from Gerhardt, who has paid special attention to this chapter up to the present (7), Schnitzler (8), Bäumlér (9), George Johnson (10), Von Ziemssen (11), Morell Mackenzie & Evans (12), Riegel (13),

Oliver (14), and Nicolas Duranty (15) must here be mentioned. The contributions of Bäumlér and George Johnson are specially important from the standpoint of principle, inasmuch as these two authors showed even at that early period of more recent investigation, that a *unilateral* affection of the *vagus* may under certain circumstances produce a *bilateral* paralysis of the vocal cords, or paralysis on the one side, and spasm on the other. Their observations have subsequently been corroborated by McCall Anderson (16), Whipham (17), and Kurz (18), whilst the cases described by Schnitzler (19) and Sommerbrodt (20), in which, according to these authors, a *unilateral* paralysis of the *recurrent* laryngeal had produced a *bilateral* paralysis of the vocal cords, were erroneously interpreted, as I have shown (21) in connection with Sommerbrodt's case.

In the period between 1860 and 1870 a number of special text-books were published, in all of which the paralyses of the larynx are dealt with, in more or less detail.

The first place probably belongs to the work of Türck (22), which in addition to many well-illustrated and accurately observed cases, contains an attempt at an etiological classification and precise subdivision of the several forms of paralysis.

In 1866 there appeared a little work by Morell Mackenzie (23) on motor disturbances of the larynx. It is distinguished by its casuistic wealth, and deals more specially with the method of direct electrification of the vocal cords by means of the author's laryngeal electrode, but suffers from the mistake that organic paralysis of the trunks of the laryngeal nerves is throughout confounded with functional paralysis of the adductors of the vocal cords. The same error deprives the industrious chapter on paralyses of the larynx in J. Solis Cohen's (24) great text book of much of its value.

To the same period belong the experimental contributions of Philip Schech (25) and G. Schmidt (26); the former will always remain a laryngological classic, inasmuch as it contains, in addition to its bibliographical treatment, numerous carefully conducted and critically discussed experiments on the functions of the individual nerves and muscles of the larynx, and on the bearing of these physiological data upon the pathology of laryngeal paralysis. We may note that Schech already thinks of the existence of a reflex-tonus of the posterior crico-arytænoid muscle, when discussing the position of the vocal cords during quiet respiration—a point inexplicable at the time, and to which we shall presently return.

From about 1876 the study of laryngeal paralyses was directed to an interesting form, characterised by loud sonorous inspiration, severe and purely inspiratory dyspnœa, perfectly preserved voice and free expiration. I speak of that affection, which up to Krause's paper, which will be fully discussed hereafter, was generally spoken of as "bilateral paralysis of the abductors of the vocal cords." It is true that already, at an earlier period, such cases had been reported; the first by Gerhardt in 1863 (*loc. cit.*, pp. 268 and 269); the next—a doubtful one—by Türk (*loc. cit.*, p. 461); the third by Morell Mackenzie and Hughlings Jackson (27); the fourth also by Mackenzie (28); the fifth by Riegel (29); yet it was only the classical paper on "Respiratory Paralyses" (30), contributed by the last named author which in 1876 attracted the general attention of observers.

But even in this paper, as well as in other contributions to the question (Penzoldt (31), von Ziemssen (32), Burow (33), and Bosworth (34), no attempt was made to trace to general laws the curious fact that in affections, which apparently concern the *entire* centres or trunks of the motor nerves of the larynx, only the *abductors* of the vocal cords or the nerve twigs which supply these muscles are found to suffer. On the contrary all these writers only sought to explain by some hypothesis or other the apparent pathological curiosity, apart from any other correlated instances.

Only at a later period was it found that the phenomenon is but an example of a definite law, and this fact was discovered in the following manner:—

In the years 1876 to 1878 I had the opportunity of observing at the Hospital for Diseases of the Throat a series of six cases of bilateral paralysis of the glottis-openers which I described in April, 1878, in a paper (35), read before the Clinical Society of London. In some of these cases the cause of the disease could not be traced definitely; in others, however, the clinical or *post-mortem* investigation proved that central or peripheral affections had been present, which evidently had involved the *entire* centres or trunks of the motor laryngeal nerves. Nevertheless in these cases the appearances of isolated paralysis only of the glottis-openers had been present during life; after death the posterior crico-arytænoid muscles only, or at any rate to a much higher degree than the glottis-closers, were found to have undergone fatty degeneration and atrophy, whilst in the nerves some normal nerve fibres were found among numerous degenerated ones.

Whilst I was studying these cases of *bilateral* paralysis of

the glottis-openers an even larger number of cases came under my observation, in which the same phenomenon was observed *unilaterally*, *i.e.*, in which isolated paralysis of one posterior crico-arytænoid muscle was seen combined with lesions of the entire nerve trunk or its medullary centre.

The *constancy* of this phenomenon necessarily attracted my attention, for whilst the exclusive appearance of paralysis of the *abductors* was so frequently seen in organic lesions of the centres or of the nerve trunks, I did not once see the reverse occur under similar circumstances, *i.e.*, isolated paralysis of the *adductors* of the vocal cords.

At that time we all were entirely under the influence of the doctrine which had gradually become developed in the course of the preceding 18 years, that, whilst in central or peripheral lesions of the entire motor apparatus of the larynx individual muscles *could* be *exclusively* affected, yet it depended more or less upon *accidental* circumstances of the individual case *which* of these muscles would be paralysed. This doctrine was advocated as late as 1876, in Von Ziemssen's *Cyclopædia* (36). My own experiences, however, as shown above, seemed to contradict it so decidedly that I instituted a thorough search through laryngological literature, in order to find out whether these experiences were *exceptional*, or whether indeed the abductors of the vocal cords and the nerve twigs leading to them were really prone to much earlier and more frequent affection than the adductors, in progressive organic lesions of the entire centres and trunks of the motor laryngeal nerves.

The result of this search greatly surprised me. For according to the current doctrine the number of cases in which an initial incomplete paralysis of the recurrent laryngeal nerve manifested itself by the symptoms of a paralysis of the *abductors*, ought to have been equal, or nearly equal to that of the cases in which the adductors were chiefly affected. Now, it resulted from my enquiry, that *not a single* really valid case, at any rate not one authenticated by *post-mortem* examination, could be found in the whole domain of laryngological literature, in which organic progressive lesions of the nuclei or trunks of the motor nerves of the larynx had manifested itself by an initial paralysis of the *adductors*. On the other hand I found quite a large series of recorded cases, in which under similar conditions in the commencement of the disease and often up to the death of the patient, the symptoms of paralysis of the *abductors* had manifested themselves and in which after death atrophy and degenera-

tion of the posterior crico-arytænoid muscles only (or at any rate more marked than in the adductors) had been present. In other words, my own observations were not, as I had originally believed, *exceptional*, but conformed themselves to the *generalised* sum of previous experiences.

Even before I had arrived at this final result, an opportunity offered itself of publishing the experience I had so far gained. As I have already stated on a previous occasion (37), in Sir Morell Mackenzie's work, which was just then about to be published, and of which I edited a simultaneous German edition, the author had adopted von Ziemssen's view, that it depended more or less upon *accidental* circumstances of the individual case as to *which* set of fibres of the motor laryngeal nerves would first be paralysed. It was only after I had communicated the results I had so far obtained to Sir Morell, that the paragraphs dealing with the subject were altered by him in the sense of acknowledging and emphasizing the greater frequency of initial paralysis of the *abductors* (38).

The footnote (p. 574) which I added to his statement in the German edition incontestably proves that I had already at that time clearly recognised the *importance* and *bearings* of the whole question.

This footnote runs as follows:—

"From the above it will be seen that the author assumes a differentiation to exist in the centre itself of the nerve twigs supplying the individual muscles of the larynx. The German editor agrees with this view on theoretical grounds, but wishes to draw attention already here to the fact that even such a hypothesis does not suffice to explain a most remarkable phenomenon, viz., that in many cases in which *central* or *peripheral* lesions unquestionably affect the whole nerve (most frequently in cases of pressure upon the entire trunk of the recurrent laryngeal in cases of aneurism of the aorta) the symptoms of paralysis of the glottis-openers only are seen during life. Whilst this has been observed by the author as well as by the editor in a considerable series of cases (several of them will be fully related further on), *the latter knows of no case* in which, after paralysis during life of one or several of the adductors *post mortem examination has shown disease of the entire nerve trunk*. This proclivity of the fibres going to the abductors to be affected exclusively or long before the others, is very strange."

I hope I need make no excuse for recapitulating here the stages in the development of this topic. I have been so frequently

asked how I had arrived at formulating the law that I must conclude that my previous descriptions of its origin have not been exhaustive enough; moreover, this historical sketch will enable every unbiassed reader to form his own opinion on the unpleasant and long continued discussions between Professor O. Rosenbach and myself as to the question of priority.

Before the publication of Mackenzie's work and of my own first special paper on this point, Rosenbach had observed a case of bilateral paralysis of the vocal cords due to carcinoma of the œsophagus, originally presenting a paralysis of the glottis-openers, which gradually passed over into total paralysis of the recurrent laryngeal nerves, and on the strength of this single observation had formulated this conclusion (39): "*Above all, the fact must be registered that in compression of the trunk of the recurrent, the function of the abductors suffers first, and that the adductors are implicated only later on.*" Now this is undoubtedly correct, as shown by experience; and it is acknowledged that Rosenbach's publication appeared a little before my own first special contribution to this question. But with every effort at preserving an objective attitude, I cannot characterise Rosenbach's statement otherwise than as a mere *contention*, most venturesome at the time, and which, luckily for the author, ulterior events proved to have been correct. I maintain most definitely:—

1. That his *generalisation from one single case* was not justified;
2. That his statement refers only to *peripheral paralysis by pressure*, not to *central affections*.
3. That Rosenbach has adduced *no real proof of the general validity* of his statement, even for this pressure paralysis.

For further details the interested reader is referred to the controversy (40).

The first special paper on this question I published appeared under the title, "Clinical remarks on the proclivity of the abductor fibres of the recurrent laryngeal nerve to become affected sooner than the adductor fibres, or even exclusively, in cases of undoubted central or peripheral injury or disease of the roots or trunks of the pneumogastric, spinal accessory or recurrent nerves," in 1881, in the *American Archives of Laryngology*, (41) since defunct.

This paper culminated in the *establishment of the fact*—it will presently be seen why I lay special stress upon the italicised words—that in the various progressive *organic* lesions of the motor laryngeal nerves, when the lesion of the affected nerve

had not been from the very beginning equivalent to its complete transverse section, symptoms of paralysis of the *glottis-opener* only had, without exception, been observed at the commencement and often up to the death of the patient; and that after death isolated, or at any rate more advanced degeneration and atrophy of the posterior crico-arytænoid muscle had been found; whilst not a single case had been reported in which under similar circumstances the *glottis-closers* had been the muscles first affected. On the other hand, it was remarkable that in all so-called *functional* motor neuroses of the larynx the *adductors* had almost always, if not without exception, been the only muscles affected.

An explanation of these facts *was not* attempted in the paper referred to. It is true that different hypotheses were ventilated which could possibly have contributed to explain the curious fact, and—what I wish to mention more especially in view of Krause's hypothesis—I *myself* already then discussed a very obvious possibility, viz., that the phenomena observed during life might *not* belong to a primary *paralysis* of the *abductors*, followed by paralytic contracture of their antagonists, but to a *spasm* of *all the muscles of the larynx*, with preponderance of the adductors (43). Any such explanation, however, was absolutely rejected in my paper, inasmuch as neither the existence of a spasm lasting for months and even years, in consequence of direct compression of a motor nerve by a slowly growing tumour *had any analogy in the rest of pathology*, nor above all could the important fact be reconciled with this theory that in these cases the abductor muscles only, or at any rate chiefly, are found, *post-mortem, degenerated and atrophic*.

Similarly Morell Mackenzie's (44) hypothesis, viz., that the abductor filaments might possibly be more superficially situated than the adductor filaments, or that the latter might be reinforced from the superior laryngeal nerve, as well as Bosworth's theory of the uniformly *central* origin of paralysis of the abductors, were rejected as untenable, and the conclusion of the paragraph in my paper runs as follows:—

“However, all these hypotheses are open to certain objections, and none of them seems to me to offer a really satisfactory and plausible explanation. But at the same time I do not believe that an *explanation* is the thing we want at present.”

“In the face of the comparative novelty of, and of all the difficulties connected with, this question, I would rather believe that the time has not yet come when we can venture to give an *explanation*, but that before all by numerous further contributions,

the *fact* should receive further corroborative evidence, that there is a proclivity of the *abductor* fibres of the recurrent nerve to succumb to pathological influences affecting the roots and trunks of the motor nerves of the larynx."

The fate of this paper has been somewhat peculiar. I do, of course, not complain that it shared the common lot of almost all publications which bring anything new, and that it was at first corroborated from different quarters, and afterwards attacked from others (45). But what is really curious is, that just those two points which I myself look upon—if the word "merit" is here applicable—as the two most meritorious ones of the whole paper, have given rise to misunderstandings and controversies which not only have formed a literature of their own, but continue *up to the present hour*, and so render any agreement upon the whole question extremely difficult. It is therefore absolutely necessary that these two points should at last be definitely cleared up.

The first of them concerns the *title* of my paper, or rather the word "proclivity" used in it. If this word had given rise *from the beginning* to misunderstandings as to what I meant, I should of course, attribute the fault to myself. Even now, I will by no means contend that the word, especially in the German translation, has been a very happily chosen one.

If I had spoken of the greater "vulnerability" or "liability" of the abductor twigs and muscles, the misunderstandings which have clustered and are still clustering round the expression, might possibly have been either entirely avoided, or at any rate, been more limited in number. But the strange thing is, that *during the first three years* after the publication of my paper *everybody* understood the expression exactly in the sense in which I myself intended to use it! The proceedings of the Laryngological Section of the 9th International Congress in 1881, in which the whole question was fully discussed by the most competent judges (46), the first confirmations of my observation [see footnote 45], nay, even the first *attacks* on the general validity of my statements,—all show that at first at any rate everybody knew exactly that I meant to signify by the word "proclivity" nothing else but the greater "vulnerability." It was only since 1884 that a change occurred in a number of contributions to this question, as if a sudden belief had arisen that this expression involved some entirely new mystic element; and since then several authors, when they speak of my paper, never quote the word "proclivity" otherwise than between inverted commas.

How I came to use this term is evident from my foregoing description. I had, when I first mentioned the fact in the German edition of Mackenzie's work, used the expression "this greater proclivity (*Geneigtheit*) of the abductors, to become affected long before the adductors, often even exclusively, is very curious indeed." When about one year afterwards I wrote my first paper on this question in English, nothing was more natural than that I should have simply translated the German expression "*Geneigtheit*," which I had originally used.

Thus originated the expression "proclivity," and that I should have used in the title of my paper such a still restricting expression, is to be attributed only to a *degree of caution* which indeed I should like to put to my account as a sort of small merit.

I had after ample personal experience, and after a search through the whole laryngological literature, arrived at the result, that in the cases in question so far *without exception* the abductors had been found affected exclusively, or at any rate, more so than the adductors; it would then have been natural enough, as Rosenbach indeed has done *after one single observation*, to formulate this result as a *pathological law*, and to give to my paper such a title as "On the law determining the order in which the individual fibres of the recurrent laryngeal nerve successively are affected in progressive organic lesions of the motor laryngeal nerves." But it must not be forgotten that all this, at the time when my paper was published, was not merely *new*, but was in *direct opposition to the generally accepted doctrines!* I did not wish to incur the possible risk of seeing a few weeks after the publication of my paper, the law I had just proclaimed impugned on the strength of an unimpeachable case. Thus I preferred to be still cautious, and to speak only of a "proclivity," even when the idea of the "law" was before my own mind. That this chain of thought is not a subsequent interpretation, but actually guided me already in 1881, is shown by the following sentence of my first paper (47); "the laryngological literature is already so large that it is not impossible that a few cases of this sort (*viz.*, of initial paralysis of the *adductors*) might have been described, of which I have no knowledge;" and from the fact that to this sentence I make the following foot-note: "I earnestly hope that this paper will serve in eliciting contributions corroborating or refuting, as the case may be, my statements, from those who have had and who have the opportunity of seeing cases in point."

After this full explanation of my chain of thought I trust we may hear no more about the "mysticism" with which my language has been charged.

Still more strange mistakes, however, are made concerning the second point, to which reference was made just now.

From the quotation of the conclusion of my paper it is seen that no *attempt* had been made in it at an original explanation of the newly found phenomena; on the contrary it had been explicitly stated that such an explanation for the time being was not urgently required.

The cause of this self-restriction was simply that the previous attempts at explanation mentioned in the paper were obviously insufficient. A satisfactory hypothesis was prevented by a fact hitherto only casually referred to by Schech (*loc. cit.*), to which my attention had been directed whilst studying the question of abductor paralysis, when I was compelled to observe accurately the position of the vocal cords during quiet respiration. I found that the usual description of the "rhythmical excursions" of the vocal cords during normal respiration *did not apply in the great majority of cases* of healthy adults, in about four fifths of whom the glottis forms an isosceles triangle, which during inspiration is but slightly, if at all, increased, and in expiration is just as little diminished in size. Exact measurements by means of graduated mirrors showed that this triangle was *two-and-a-half to three times larger* than that formed by the glottis *after death* ("cadaveric position" of the vocal cords.) This greater width of the glottis during life, which was not easily explicable *primâ facie*, certainly seemed to point to a *greater strength of the abductors* in comparison with the adductors, and the fact that these apparently *stronger* muscles should more easily succumb than the adductors to organic lesions became the more mysterious. Since then I have persevered in giving attention to this question, and have supplied a solution, I trust, of a satisfactory nature in a paper (48) which I brought before the Royal Society in 1890. In suggesting that the position of the cords during quiet respiration is the consequence of a *reflex tonus* of the posterior crico-arytænoid muscles, I find myself in accord with Krause, who simultaneously with, and independently of me, discussed this question in 1884, on the occasion of the eighth International Medical Congress at Copenhagen (49).

At the time, however, when my first paper on abductor paralysis was published, I had not yet arrived at a full comprehension of the conditions in question, and for that reason *refrained from any attempt at explanation*. That I did so *intentionally* is proved—quite apart from the quotations given above—by the following passage of my second paper on the subject

published in 1883 (50). "An explanation of my own of the fact which here was proven for the first time, I do not give, for the good reason that I do not know of one. Thus much was clear to me, that every attempt to explain the central and the peripheral cases by *different* theories, whilst in both cause and effect were identical, was fundamentally hopeless; to the only common and plausible explanation however, which was before my mind, a certain physiological fact did, at the time, appear to oppose itself as an insurmountable obstacle. Further study of this fact has *now*, I believe, enabled me to understand the real meaning of the pathological phenomenon, and I hope to give the explanation in the course of the forthcoming winter."

These repeated statements seem to me so very unambiguous that it is really quite unintelligible to me how, in spite of them, a number of authors of different nations—and in spite of repeated protests on my own part—have persisted in speaking of "Semon's theory." One can hardly open a paper which deals with the question of abductor paralysis, without encountering such expressions as: "La théorie de Semon," "die Semon'sche Hypothese," "teorema Semon," "Semon's hypothesis," &c., &c. One author summarises my views as follows: "Semon seeks the cause of this curious phenomenon in a proclivity of the abductors to become diseased." Another writer constitutes himself the advocate of Nature, which he thinks I have most seriously offended by attributing to it a "proclivity to attack one of the most vital muscles of the human system." A third admits, it is true, that I had directly stated that I would not attempt to explain the phenomenon, but at the same time outdoes his predecessors by declaring that I had "unintentionally given an explanation by registering the morbid process through the title of my paper under one exclusive cause."

A similar obstinacy in forcing upon an author various theories and explanations, which he not only has never brought forward, but against which he has *expressly protested* in the plainest terms, constitutes a rather exceptional phenomenon in medical literature. It can only be surmised that the authors who avail themselves of such expressions in discussing my papers have either not read them in the original, and have derived their knowledge only from inaccurate or incomplete abstracts, or that their ideas of what constitutes a "theory" and what a "fact" are somewhat hazy.

Anyhow my reply is as follows:—If during life in a case in which the *whole* of the nerve trunk or the *whole* centres of the motor laryngeal nerves on one or both sides are organically

affected, I observe the symptoms of paralysis of one or of both the glottis-openers, and this under circumstances which *everywhere else in the whole range of pathology* would induce every observer to think of *paralysis* only and not of *spasm*; and if *post-mortem* I find the posterior crico-arytænoid muscles shrunk to hardly recognisable discoloured bundles, whilst the adductors, which are supplied by the same nerve, are either normal or at any rate much less degenerated and atrophied, I then establish a "fact," but I do not evolve a "theory," by stating that in this case the abductors have given way before their antagonists. If this occurs not only in a certain number of cases, but *without exception* in all cases of various *organic lesions progressively affecting* the whole centres or trunks of the motor laryngeal nerves, then the law necessarily follows that the nerve fibres of the motor laryngeal nerves going to the abductors, and these muscles themselves, succumb to organic affections sooner than, or even exclusively of, the adductors.

This is what I have stated, *this* is what up to the present day has remained a positive fact, and to speak of it as a "theory" or a "hypothesis" is inadmissible.

My language may appear too emphatic to those who are not conversant with the literature of this question, and it may possibly be objected that this interferes with the objectivity of a historical description. I felt it, however, to be my duty to give such a description of the development of these questions as will, I hope, be useful to future investigators; and to refer in plain language in the hope of removing them, if possible, to the truly incredible (51) misconceptions, which up to this day stand in the way of an agreement between the adherents of the different views.

My last-named paper formed the starting-point, so to say, of a new branch of laryngological literature, which, scanty from 1881 to 1883, increasing very considerably during the following three years, attained its maximal development about the year 1886, and which has since grown so as now to fill nearly three large volumes.

The character of these contributions varies very considerably. In the first place numerous casuistic communications have been and are being contributed, confirming the fact stated by me. Amongst these I may mention the papers by Schäffer (52), Jonquière (53), Kothe (54), Ziegelmeyer (55), M'Bride (56), Dehio (57), Bristowe (58), de Havilland Hall (59), Garrod (60), Percy Kidd (61), Gordon Holmes (62), J. Israel, Remak, B. Fraenkel (63), Aronsohn (64), Schech (65), Landgraf (66), Martius (67), Dreyfuss (68), Neumann (69). To the same category belong also

the commentaries of Riegel (70), Massei (71), Buzzard (72), Ormerod (73), Ferrier (74), Gottstein (75), Ariza (76), acknowledging the correctness of the fact. Some of these authors (Buzzard and Ormerod) endeavour to utilise and explain analogous pathological occurrences in other organs, as for instance the order of events in the disablement of the individual muscles of the eye.

We now come to the interesting observations by Jeanseline and Lermoyez (77), on the dead bodies of cholera patients, showing that of all the laryngeal muscles supplied by the recurrent laryngeal nerve, the posterior crico-arytænoids are the first to lose their electric excitability after death—an observation which Horsley and I (78) confirmed in various species of animals. The fact also, corroborated by Fleming (79) and Clarke (80), that in the so-called roaring of horses, which Fleming attempts to trace mainly to the pressure upon the left recurrent laryngeal of enlarged lymphatic glands, situated near the arch of the aorta, the left abductor is always found to be degenerated alone or chiefly so, points in the same direction.

In opposition to this evidence, attacks upon the general validity of my statements were made at first in the form of publication of isolated cases, which in the opinion of their authors appeared to prove that in organic progressive disease of the motor laryngeal nerves the adductors occasionally become affected first. To this category belong the papers by Schnitzler (81), S. Solis-Cohen (82), J. Solis-Cohen (83), and Charazac (84). It was easy for me to show in my second (85) paper, published in 1883, that the cases published by Schnitzler and J. Solis-Cohen do not in the least prove the assertions made by their authors; and the same may be said of the more recent cases published by S. Solis-Cohen and Charazac. The first of these is in all probability of the nature of a *mechanical* disturbance of mobility; the second most likely of a *local* disablement of one or the other of the adductors. These cases have not been quoted by anybody but by their authors as invalidating my law. The very interesting cases published by Remak (86), (Bilateral paralysis of the spinal accessory nerves), Cartaz (87), (Cases of laryngeal paralysis of central origin), Seifert (88), (Saturnine paralysis of the larynx) can be cited neither for nor against my statements.

With reference therefore to any attempts to refute my law by *casuistic* proofs, I must now emphasize the following statement:—

Whilst a large number of cases of progressive organic disease of the motor laryngeal nerves have been published, in which the

abductors have been shown to have undergone, either alone or at any rate much more than the adductors, fatty degeneration and atrophy (89), not a single specimen has been hitherto demonstrated, which, under similar conditions, exhibited the opposite order of events in the development of degenerative changes in the individual laryngeal muscles !

All such attacks made so far upon the law exclusively rest upon *clinical* observations, which are either incomplete or are capable of an interpretation different from that adopted by their authors.

The theoretical doubts of Gouguenheim (90) and Paul Koch (91) need only be referred to in passing. Gouguenheim and his followers, who consider the median position of the affected vocal cord to be a spastic phenomenon, believe that the latter is due in a large number of cases to pressure of the peritracheo-laryngeal ganglia, described by himself and Leval-Piquet (92), upon the recurrent laryngeal nerve.

Shortly after the commencement of the period of casuistic corroboration and attacks, the first attempts at explanation were made. Curiously different hypotheses were brought forward in order to render more intelligible the clinical phenomenon. Thus Gowers (93) thinks that it may possibly be a consequence of the advantage with which the most important adductor, the lateral crico-arytænoid muscle, works in comparison with the abductor (in so far as the former goes in a right angle, the latter in a very acute angle towards the muscular process of the arytænoid cartilage), which renders the adductors capable of longer resisting disabling influences which affect the whole nerve trunk.

W. Robinson (94) attempts to explain the fatty degeneration and atrophy of the abductors, which had been observed by myself (95) in a case of goitre, by a supposed disturbance of their vascular supply, inasmuch as the posterior crico-arytænoid muscles are supplied by the inferior laryngeal arteries which had probably been compressed in this case; whilst the immunity of the adductors was, he thought, to be explained by assuming that the superior laryngeal arteries which supply them had not been injured by the pathological process.

The third hypothesis is due to Cohen-Tervært (96), and is founded upon the results of Exner's (97) well-known paper on the innervation of the larynx. It may here be mentioned that I shall not further refer in this historical sketch to Exner's paper, nor to the studies of his pupils, Mandelstamm (98) and Weinzweig (99), interesting and meritorious as they are, because there is, so far-

as I can see, no direct connection between them and our present subject, and because they have led to no results of any practical value.

The distribution of the motor nerves to the individual laryngeal muscles, as stated by Exner, and the anomalies of nerve distribution occurring everywhere, led Tervært to imagine that in the cases now discussed, the nerve-supply of the laryngeal muscles deviated from the rule in so far that the abductor is innervated by the recurrent laryngeal, to the exclusion of the superior laryngeal nerve; whilst the other muscles received more motor fibres from the superior laryngeal nerve. This hypothesis, which is based upon the assumption that in cases of abductor paralysis in which the entire nerve trunk is affected, the isolated paralysis of the posterior crico-arytænoid muscles must necessarily persist to the very last, collapses before the simple fact that in many cases, the initial abductor paralysis finally evolves into complete recurrent paralysis (100).

Further, if this hypothesis were correct, in the cases in question the external thyreo-arytænoid muscle ought always to be found to be at least as much atrophic and degenerated as the posticus. For, whilst, according to Exner, the latter is supplied only *predominantly* by the recurrent laryngeal, according to the same authority, the external thyreo-arytænoid is "usually *exclusively*" innervated by the recurrent. Such a condition of things, however, has not been found in any of the numerous cases in which abductor paralysis has come under observation. Many more objections could be raised against Tervært's hypothesis, but these two suffice to show its untenability.

The complex far-fetched and not very intelligible theories brought forward by S. Solis-Cohen (101) and Jelenffy (102), the mere statement of which would demand a disproportionate amount of space are so little founded upon facts, that they are only alluded to here for the sake of completeness.

Tissier's hypothesis is equally shadowy; the recurrent laryngeal is not a simple nerve, but contains (i.) vagus fibres, (ii.) spinal fibres. The former are said to be present only in small numbers in the recurrent laryngeal, and their excitability is supposed to be lost sooner than that of the spinal fibres. In compression of the nerve or in peripheral neuritis, the vagus fibres are thus the first to be exhausted, the spinal fibres retaining their function, which being phonatory allows the approximation of the vocal cords.

A very curious hypothesis has been quite recently proposed

by Wagner (104) of Halle. This author found that after unilateral as well as after bilateral section of the recurrent laryngeal nerves in cats, and in the very few dogs experimented upon, the corresponding vocal cords assumed a median position and immobility which lasted for a few days and then passed over into the so-called "cadaveric" position. He explains the median position of the vocal cord or cords under such circumstances as being due to the action of the *crico-thyroid muscle only*.

This explanation appears so simple that it would be extraordinary if no previous observer had found it, provided, of course, that the author's facts are correct.

Now it would appear that Wagner is not aware that section of the recurrent laryngeal or laryngeals has been performed before him by *numerous* experimenters; at least he does not allude anywhere to the analogous experiments of his predecessors, nor does he attempt to explain the differences between his own and their results. Had he studied the literature of the subject he would have seen that experiments concerning this question have been made since the beginning of the present century by Legallois, Read, Longet, Traube, Rosenthal, Dalton, Georg Schmidt, Schech, Steiner, Vierordt, jun., Horsley and myself, &c., and that although all these observers agree that after section of the recurrent laryngeals the glottis becomes much narrower than it had been previously, yet that there are very great differences as to the *degree* of the narrowing, according to species, age and individual peculiarities of the animals experimented upon. While certain species, such as cats and horses, not only in the first days after birth, but even when adult, are suffocated by the section of the recurrent laryngeals, other species, notably dogs, suffer less and less, in proportion to their age, so that, whilst they die when operated upon a few days after birth, dyspnœa only occurs on exertion when they are operated upon when adult. Again, other species, such as rabbits and guinea pigs, are not nearly so much inconvenienced when operated upon, even at a very early period of their existence. The reason of these differences is well explained by Legallois, the first thorough worker in this field (*Nouveaux éléments de physiologie*, 2 Edit., vol. xi., p. 326 et seq.), as follows:—

"It rests upon the fact that, in proportion to the capacity of the lungs, the opening of the glottis in animals of the same age is greater in one species than in another, and still greater in the adult than at the moment of birth in the same species, as already stated by M. Richerand, in the human species. Or, assuming

that the form of the glottis on the whole is similar in these diverse animals, inasmuch as the areas of the smaller figures are to each other as the squares of the homologous dimensions, it is seen that a narrowing of the same kind of the glottic opening must intercept the passage of air in very different degrees."

From this quotation alone it is evident that there cannot be in all cases bilateral median position of the vocal cords, *i.e.*, *complete closure* of the glottis, after section of the recurrent laryngeals, for, if such were the case it would be impossible to understand how any animal thus operated upon could have *survived* the operation. Yet Legallois and many of his followers have kept such animals alive for long periods. Moreover, several of the experimenters, who directly *inspected* the position of the vocal cords after section of the recurrences, *e.g.*, Traube, Schech, Steiner, Horsley and myself, Katzenstein, have actually *seen* the corresponding vocal cord recede into the *cadaveric* position at the *very moment* its recurrent laryngeal nerve was cut.

This is entirely at variance with Wagner's results, who, without exception, found in his unilateral sections, the median position of the vocal cord to last up to six days. Yet he does not as much as mention this grave discrepancy between him and his predecessors.

Now it appears to me to be the *duty* of any observer who enters upon a field well trodden by previous investigators to examine their *facts*, if not their *theories*, and either to refute them or to reconcile with them any new hypothesis which he, as the result of his own experiments, may bring forward. But he has no right, I think, to leave such facts simply out of court, and to give an entirely erroneous impression of the actual state of matters to readers who may not be familiar with the subject. This is what Wagner has actually done.

Since his publication Katzenstein, of Berlin, has made a series of very careful and varied control experiments, and has been unable to corroborate Wagner's statements; his own results, which are given in Virchow's Archiv, vol. 128, entirely agreeing with those of the older observers.

This being so I shall confine myself to pointing out a few other obvious objections which might be raised against Wagner's *interpretation*, assuming for a moment, what I do not admit, that his *facts* are correct.

1. Supposing that after mere section of the recurrent laryngeal nerve the vocal cord at once *invariably* went, as he describes it, into the middle line, and that this was due, as he thinks, to the

action of the crico-thyroid muscle; it necessarily follows that the position of the vocal cords, which we see in quiet respiration in man, is not merely a result of a reflex-tonus of the abductors (as would appear from my paper in the Proceedings of the Royal Society, 1891), or even of a simultaneous innervation of the abductors and adductors, with preponderance of the former, but that the *crico-thyroid* also must play an important rôle in the production of this position. Surely the author cannot believe for a moment that this muscle only *began* to exercise a constant activity at the very moment at which the recurrent laryngeal was cut? Such a belief appears so inherently improbable as to be not worth discussing. Supposing, however, on the other hand, that the crico-thyroid muscle in man is *always* engaged in maintaining the position of the vocal cords during quiet respiration, and in counter-balancing, to a certain degree, the action of the posterior crico-arytænoid muscle (as we must conclude from Wagner's results from recurrent section), it would again follow that, if the *crico-thyroid* itself were disabled, the glottis in quiet respiration ought to be *wider* than it is under normal conditions. Now I am not acquainted with a single *pathological* observation pointing this way, whilst in *experiments* in which the crico-thyroid has been cut in animals (*e.g.*, by Schech and Steiner), no appreciable change in the width of the glottis was observed. Neither did Katzenstein obtain any widening of the glottis after dividing the external branch of the superior laryngeal nerve which supplies the motor filaments to the crico-thyroid muscle. Thus neither experiment nor pathological observation give the least support to Wagner's contention, whilst he himself never once enters upon the all-important point I have just raised.

2. Wagner, in the whole of his three extensive papers on the subject never condescends to investigate the cause of the exclusive or more developed degeneration and atrophy of the posterior crico-arytænoid muscle in cases in which median position of the vocal cord from progressive organic paralysis of the recurrent laryngeal nerve had existed during life. This point he does not even mention, though he himself reports in several of his experiments that the electric excitability of the posterior crico-arytænoid was lost after death previous to that of the adductors.

3. Finally, no explanation whatsoever is given of the fact that the median position in man may and does last for many years, whilst its maximum duration in Wagner's experiments was six days.

More might be said about the defects of the author's bibliographical researches, about the probable sources of fallacy in his experimental methods, about his confident generalisation of the results of a few experiments, and about his own far-fetched explanations; but what we have just seen will be enough to show that in spite of their apparent simplicity, Wagner's views are not likely to meet with general acceptance.

Having so far disposed of the theoretical, we now reach the anatomical and histological contributions. Grützner (105) appears to be inclined to look upon the adductors as "white," and the abductors as "red" muscles; Simanowsky (106), who worked in Grützner's laboratory, directly characterizes them as such. These results are opposed to the fact which may now be considered as certain, viz., that the abductors first succumb to any organic progressive lesions of the whole nerve, and to the physiological experiments which have been made by Horsley and myself (107); and I must content myself here with pointing out these as yet unsolved contradictions. If in reality the abductors belonged to the class of red, the adductors to the class of white muscles, we should have expected, in accordance with what we know of the biological properties of these tissues, the abductors to resist organic pathological influences longer than the adductors.

We shall presently see that this bio-chemical question has to be diligently studied, as it probably contains the final explanation of the different resistance of the glottis-openers and glottis-closers to progressive nerve-lesions.

Finally, we must here mention the experiments concerning the excitability of the recurrent laryngeal nerve, made by Franklin H. Hooper (108) and Frank Donaldson, Junr. Hooper's remarks in the discussion which followed the reading of his paper (*Transactions of the American Laryngological Association*, 1885, pp. 23 et seq.), probably contain the most violent attacks ever made upon the fact of the greater vulnerability of the abductors. But they are not based upon valid data, nor do they in the least disprove the fact stated by me. The real meaning of the important discovery which Hooper accidentally made in the course of his experiments, viz., that ether possesses a peripheral and differential action upon the laryngeal muscles, was not duly appreciated by the discoverer himself, and was recognized subsequently and explained by Horsley and myself (110).

In the first of his papers Donaldson directly attacks the fact of the greater vulnerability of the abductor fibres; in the second, however, he fully acknowledges it. These contributions possess

permanent value as demonstrating the greater excitability of the abductors when weak electric currents are used. Both the last-named authors have followed up their original observations by further interesting excitation experiments on the recurrent laryngeal nerve; but their later papers do not appear to shed any special light upon laryngeal paralyses.

We thus see that there has been no want of diversity among the various hypotheses propounded in explanation of the phenomenon of the greater abductor vulnerability, although none of them has succeeded in gaining general acceptance. The majority have only raised a passing interest, the rest being still-born.

The last hypothesis to be now mentioned, however, has rallied a number of adherents, whilst others are maintaining an expectant attitude in the fight between its partisans and those of the older view. I am alluding to Krause's contracture hypothesis (111).

Krause started with the wish to imitate by experiment the pathological process, a sufficient explanation of which at that time had apparently been given neither by clinical observation nor by the results of autopsies, but failed in a preliminary series of experiments to produce the laryngeal phenomenon, as it were, under his eyes. He now attempted to imitate experimentally the pathological process upon which the median position of the vocal cords most frequently depends during life, viz., the pressure of a tumour upon the motor nerves of the larynx. With this view he fixed a piece of cork, by means of an indiarubber band or a violin string, softened in water, to the previously carefully isolated recurrent laryngeal nerves, which were then replaced in situ. After a few hours he first observed slight vibrating twitchings, afterwards somewhat temporary median position, and after about 24 hours, lasting median position. This median position persisted without any change for two or three days. Afterwards it passed over into complete paralysis. If the same experiment was performed on the pneumogastric nerve (it deserves special mention that Krause always operated on both sides), the vocal cords assumed permanently the quiet position of expiration.

From these experiments Krause concludes that the phenomenon produced by him must be looked upon as one of *excitation*. He attempts to prove, partly by a few clinical cases which had been previously reported, partly by theoretical considerations, that in *man* also the pathological process does not consist as hitherto assumed, of a *primary paralysis* of the posterior crico-arytænoids, followed by paralytic contracture of the antagonists,

but of a *primary* neuropathic *contracture* of all the muscles supplied by the recurrent, with preponderance of the adductors.

The above is a faithful, though concise, analysis of this important paper. The reader must for details refer to the original.

It is not surprising that Krause's theory should have found and still finds numerous adherents. It provides an intelligible explanation for the previously obscure cause of the phenomenon; it is *prima facie* very attractive, owing to its great simplicity; the few experiments on animals on which it rests, are considered by some of greater value than any series of careful clinical and pathological-anatomical observations; finally, it must undoubtedly be conceded that Krause's paper ably sustains his views.

Thus a number of laryngologists have declared themselves partisans of this theory, amongst whom are Möser (112), Gouguenheim (113), J. Solis-Cohen (114) (if I correctly understand his present views), Moure (115), and lastly Massei (116), who has declared himself a convert.

Now the weak points of Krause's theory are so obvious, that from the moment of its publication it encountered opposition. The author having first reported the results and the interpretation of his experiments to the Physiological Society of Berlin (117), submitted them again to the Laryngological Section of the eighth International Medical Congress at Copenhagen (118). In the discussion which followed his paper (*l.c.*) B. Fränkel, Boecker and myself raised objections against his views. I declared (*l.c.*) that whilst reserving my judgment concerning the general idea in support of which these experiments were adduced, the following objections at once came up to one's mind:

1. In cases of *multiple* paralyses, such as had been described by Remak and myself, is the affection of the vocal cords which is present amongst others signs, also to be looked upon as a contracture? Why amongst a number of undoubted paralyses this one contracture?
2. The occurrence of so complete a degeneration of the abductors as is found in these cases as a pure consequence of the contracture of the adductors was unintelligible.
3. It would be in direct opposition to *all pathological experience*, if organic disease of the vagus should produce lasting expiratory position of the affected vocal cord, as seen in Krause's experiments. On the contrary in all such cases, except when the paralysis was complete, one observed unilateral or, in some cases, reflexly produced *bilateral abductor paralysis*. I added that other objections presented themselves, but that considering the difficulty

of the question and of the experiments, we must wait for the full publication of Krause's paper.

This paper having been published, O. Rosenbach (119) and B. Fränkel (120) were the first to raise further objections against Krause's views. Both authors first criticised the experiments themselves, and next the interpretation of their results as a contracture. Möser and Tervært (*loc. cit.*), also brought forward various criticisms of Krause's interpretation. To Rosenbach and Möser's objections Krause replied in two short papers (121) of a polemical nature, the second of which finally cleared up the question as to *which* muscles were to be considered as being in a condition of contracture. It must be explained that up till then repeated misunderstandings had been caused by Krause's varying modes of expression. Sometimes one could not but imagine that he fancied that only the "*main bulk*" of the laryngeal muscles, *i.e.*, the adductors, were in a condition of contracture, sometimes he spoke of a contracture of *all* muscles supplied by the recurrent laryngeal. His reply to Möser finally settled matters definitely in that sense that in his opinion *all* laryngeal muscles supplied by the recurrent were contracted.

Gerhardt (122), Michael (123), and Weil (124), about the same time, published papers which took an intermediate position between the old and the new views.

My own objections against Krause's theory were brought forward as late as 1886 in a paper entitled: "Abductor Paralysis, not Adductor Contracture" (125), which was read before the Laryngological Section of the German Naturalists' Meeting at Berlin. I had considered it necessary, in view of the surprising statements which Krause had made about the position of the vocal cords in experimental compression of the pneumogastric nerves, to repeat the experiments themselves. This occupied much time. My paper forms part of a larger work, the MS. of which already in 1886 was nearly ready for the press, but which for various reasons has up to the present not been published. Of the paper itself therefore, only short abstracts in the Journal of the Naturalists' Meeting, and in a few reports of the proceedings have been published; and this is probably the reason why my arguments against the contracture hypothesis have apparently not become so generally known as my first articles and as Krause's own paper. I hope soon to publish the whole work; but will give here a full abstract of my paper, which contains my main reasons for not accepting Krause's theory; reasons which, so far as I know, have up to the present day remained entirely unrefuted.

After protesting against the validity of cases, observed *clinically* only, as counterproofs to my law, I proceeded by giving a report of several series of experiments on different species of animals undertaken by Mr. Victor Horsley and myself conjointly. From these it appeared that:—

1. The abductors are the first of all the laryngeal muscles proper to lose their excitability after death.

2. After thrusting a thread saturated with chromic acid solution through a recurrent laryngeal nerve, the animal is killed a week afterwards; the corresponding posterior crico-arytænoid muscle is the *first* to lose its excitability.

Here I noted, first, that I had not been able to corroborate, in man, Krause's statements as to the complete closure of the glottis during cadaveric rigidity; secondly, that the expression "cadaveric position of the vocal cords" was open to objections, inasmuch as the position of the cords after death varied considerably.

I then discussed at some length the sources of error attending experiments upon animals made for our purpose, and of which I adduced not less than eight, met with by ourselves. I drew from my experience the conclusion that experiments on animals can here be utilized only with the greatest precaution, and can certainly not be regarded as *decisive*.

If this were generally true as regards physiological experiments performed on the laryngeal nerves, it was still more so in the case of experiments intended to be *imitations* of usually *chronic* pathological processes. Krause's conclusions were, in my opinion, too far-reaching, mainly because he had identified the *sudden* and *intense* irritation he had experimentally produced, with the slow and *gradual* increase of irritation by chronic pathological processes. That the median position produced by him experimentally was to be interpreted as an *irritative* phenomenon, I quite admitted, although there was no need to term it a "primary neuropathic contracture" (this point was fully discussed); nor did I deny that in *a few* human cases of *acute* character a neuropathic median position of the vocal cord might have to be interpreted as an irritative phenomenon. It was undoubtedly meritorious on Krause's part to have drawn attention to this point, but I must reject such a *generalisation* as that contained in his papers. In man a *slow* destruction of the nerve almost always takes place, in which alterations of pressure, and with them irritative phenomena, *can* no doubt occur, but are *very frequently* absent. Even in cases of this kind the posterior

crico-arytænoid muscles, as shown by the result of the autopsy, succumbed first. I showed that Krause fell into self-contradiction when interpreting the atrophy of these muscles, as "atrophy due to inactivity," since according to his hypothesis *all* the muscles supplied by the recurrent laryngeal, and therefore also the abductor, were supposed to be in a condition of chronic irritation.

The following consideration, moreover, completely settled the question for me. If Krause's general conception of the nature of the processes taking place in these affections of the recurrent laryngeal nerve were correct, it would logically follow that *everywhere in the system where absolutely analogous processes occurred under absolutely analogous conditions the same result must uniformly take place*. Therefore, wherever pressure exercised by a slowly growing tumour gradually disables a motor nerve, a contracture must uniformly ensue. But we never find contracture in such cases, but *paralysis* (e.g., paralysis of the facial nerve or of the brachial plexus, crutch-paralysis, &c.)

This was decisive for me. The quotations from the works of Charcot, Erb, Eulenburg, Hitzig, Seeligmüller, by which Krause attempted to support his views were not admissible to solve the question at issue, inasmuch as they referred to entirely different conditions. These authors had themselves stated so much to me in reply to definite questions addressed to them on the subject.

Horsley's and my own experimental results with reference to the *vagus* *entirely* differed from Krause's, whilst they tallied, so far as they went, with the conditions met with in pathological cases.

Finally, if further corroboration were needed that the neuropathic median position of the vocal cord or cords was to be looked upon as the result of a primary abductor paralysis, we found it in cases of *multiple paralyses of cranial nerves*, such as had been accurately observed by M'Bride and by myself among others.

In such cases the domains of *all other* affected nerves displayed nothing but *paralytic* phenomena. It was most improbable that *irritative* phenomena should always occur in the domain of the vago-accessory, whilst in all other nerve-territories *paralytic* symptoms should be the rule.

Krause, no doubt, deserved our thanks for having emphasized the possible occurrence of irritative phenomena; and opening our eyes in *acute* instances of neuropathic median position of a vocal cord or cords, to the contingency not only of abductor paralysis but also of acutely irritative lesions, but the *general* interpretation of the phenomenon as contracture was inadmissible, and the

usual soundness of the old interpretation of the median position, as abductor paralysis was unimpaired.

In the discussion which followed my paper Krause defended his interpretation and criticised Horsley's and my own experiments, but admitted that in nerve affections phenomena of irritation as well as of paralysis could occur, and suggested that it might perhaps have been the enthusiasm of the investigator, which had made both him and myself too far exclusive, each of us saying: only one thing of the two can be true, the one excludes the other.

The result of the discussion was to the effect that *not one* of the numerous speakers who took part in it shared Krause's views; Remak, from the general neurological point of view, stated that he entirely agreed with me in thinking that such difficult problems are generally insoluble by means of rough experiments, and can only be explained by clinical and pathologico-anatomical observations, and that laryngologists, on the strength of some quotations, were not entitled to represent as constant a phenomenon in the domain of the laryngeal nerves, which was not observed in the territory of other motor or mixed nerves (120).

This discussion was, at the time when this paper was published in German (October, 1891), the last important occurrence in the question now before us. In 1890, on the occasion of the demonstration of the motor innervation of the larynx, which Horsley and I gave in a combined meeting of the Physiological, Neurological and Laryngological Sections of the Berlin International Congress, I accurately restated my views (127). No opposition was raised, and I believe that even then the opinion of the great majority of laryngologists, and of almost all neurologists was to the effect that in organic progressive lesions of the motor nerves of the larynx the abductor fibres and muscles regularly succumb first.

Since that time, however, some very important contributions have been published. In the first place Risien Russell has materially strengthened in a paper read before the Royal Society, on March 31st of the present year (*Proceedings of the Royal Society*, vol. 51), the position which I have throughout taken in this question, by showing experimentally—

1. That the abductor and adductor fibres in the recurrent laryngeal nerve are collected into several bundles, the one distinct from the other, and each preserving an independent course throughout the nerve trunk to its termination in the muscle or muscles which it supplies with motor innervation.

2. That when the abductor and adductor fibres are exposed in the *living* animal to the dry influence of the air under exactly similar circumstances, *the abductors lose their power of conducting electrical impulses very much more rapidly than the adductors; in other words, that they are more prone to succumb than are the adductors.*

3. That it is possible to trace anatomically the abductor and adductor fibres throughout the whole length of the recurrent laryngeal nerve to their termination in the one or other group of laryngeal muscles, and that these fibres appear to bear a fixed relationship to each other throughout their course, the abductors being situated on the *inner* side of that nerve or next to the trachea, while the adductors are on the outer side.

4. That it is possible to accurately separate these two sets of fibres in the nerve trunk.

5. That the bundle of nerve fibres concerned with one function may be divided without injury to that concerned with the opposite function, and that such division is followed by atrophy and degeneration of the muscles related to that function without any such changes being detectable in the muscles related to the opposite function.

These results, the actuality of which has been demonstrated by the author at a meeting of the Physiological Society, entirely bear out the position taken by me.

In justice to Dr. Onodi, of Budapest, it must be observed here that he communicated results practically identical with those obtained by Russell in the course of the discussion which followed Horsley's and my own demonstration at the International Congress of Berlin, in 1890. But, as he merely communicated the bare facts, without supporting them by satisfactory evidence, his observations could not claim the same value which Russell's careful experiments undoubtedly possess.

Another equally important and surprising contribution to that question comes from the pen of Krause. (*Berliner Klin. Wochenschrift*, No. 20, 1892). On the strength of new experiments on animals he arrives at the conclusion that the recurrent laryngeal nerve is not, as hitherto almost generally admitted, a purely motor nerve, but that it contains also centripetal fibres, and that irritation of this nerve therefore, apart from causing arrest of respiration and of the diaphragm in the expiratory position, also produces narrowing of the glottis and arrest of the vocal cords in the adducted position. This position, therefore, must, in his opinion, now be looked upon as the result of a 'reflex contracture.'

In a very trenchant paper, Burger (*Ibid.*, No. 30), has shown that this new position of Krause's is not, as this author maintains himself, a mere 'modification' of his former views, but constitutes an absolutely new hypothesis, which is additionally quite incompatible with Krause's former theory, according to which the median position of the affected vocal cord was to be looked upon as a 'primary neuropathic contracture.' Burger has repeated Krause's experiments, and has not been able to corroborate his statements concerning the existence of centripetal fibres in the recurrent laryngeal nerve—a result which years ago also Horsley and I have obtained. As Krause, in the course of his paper, admits that the posterior crico-arytænoid muscle may possess the least power of resistance of all the laryngeal muscles supplied by the recurrent nerve, Burger justly sees in this admission a corroboration of the facts for the correctness of which I have contended from the very first, and it may fairly be hoped that this long continued strife is now at last drawing to a satisfactory conclusion.

Nor are we now any longer quite in the dark as to the cause of the phenomenon.

The fact discovered by Hooper (123), controlled and explained by Horsley and myself (129), that ether has a peripheral and differential effect upon the laryngeal muscles, which can be produced only by means of the circulation; the fact that the abductor muscles die sooner than the adductors (Jeanselme and Lermoyez, Semon and Horsley, Onodi, Risien Russell); and the fact demonstrated by B. Fränkel and Gad (130), that gradual cooling of the recurrent laryngeal nerve paralyzes the crico-arytænoideus posticus sooner than the glottis closers; all these facts taken together with the clinical and pathologico-anatomical experiences concerning the earlier destruction of the abductors in progressive organic lesions imply that there exists an actual difference in the *biological* composition of the laryngeal muscles and nerve endings, whilst the fact that also in *central* (bulbar) organic affections, such as tabes, the cell groups of the abductors succumb earlier than those of the adductors, points to the probability that similar differentiations exist in the nerve-nuclei themselves. The hitherto obscure phenomenon thus finds its explanation in biological differences between the components of the laryngeal nerves and muscles. This constitutes an *addition* to our knowledge of the nervous morphology, but does not necessitate so *revolutionary* a postulate as that involved in the contracture hypothesis that the motor laryngeal nerve possesses

a *special* pathology, peculiar to itself. We formerly knew that there existed differences between the irritability and power of resistance of the sensory and those of the motor nerves; but assumed complete equality among motor nerves. Now we have also learnt that more subtle differences exist among these nerves and the physiological conditions of the muscles they supply.

Between the years 1880 to 1890 the interest in laryngeal paralyses was concentrated upon organic progressive lesions, from the vago-accessory nucleus *downwards*, and especially upon the abductor-paralyses just described. Recently, however, the question, whether central lesions situated *above the medulla*, especially cortical affections, may also produce organic laryngeal paralyses, has arisen. The pathological aspect of this question has grown out of the modern physiological studies concerning cortical localisations, and which have led to the discovery of definite areas presiding over the phonatory (volitional) functions of the larynx.

Ferrier (131), and shortly after him, Duret (132), in the course of their experiments reached the conclusion that the function of phonation must be represented in the cortex, but did not succeed in obtaining an exact localisation. Krause (133), who studied this question at H. Munk's instigation in 1883, first defined the exact locality of the phonatory area in the dog's cortex. He found that closure of the glottis (*bilateral* adduction of the vocal cords) takes place on stimulating the isthmus ("Stiel") of the præfrontal gyrus (gyrus præcrucialis, Owen). Krause's results were corroborated and extended by Horsley and myself (134). Those results of our investigations are as follows:—

1. That there is in each cerebral hemisphere an area of bilateral representation of the adductor movements of the vocal cords. (Corroboration of Krause's results).

2. That we could discover in none of the species (except cats) of animals experimented upon, an area in the cortex for the abductor movements.

3. That after complete excision of the cortical areas for adduction (stimulation of the neighbourhood of the lesion producing no more effect upon the larynx), and after allowing the wound to heal aseptically, no paralysis of the cords is observed. Further, that on subsequent excitation of the corresponding area in the opposite hemisphere a bilateral adduction of the cords is produced as completely as if the opposite area were intact.

From these facts we argued as follows: "Unilateral destruction produces no effect. There is, therefore, no such thing as unilateral paralysis of a vocal cord from lesion of a cerebral hemisphere, as has been asserted. The truth of our statement is made evident by the fact that motor aphasia is not identical with aphonia. Moreover the observation by one of us (F.S.) of several persons with complete aphasia and right hemiplegia only a few hours after an apoplectic stroke, has conclusively shown that in such instances the movements of the vocal cords are perfectly preserved."

Mott (135a) and Sherrington (135b) corroborated these conclusions, but François Franck (136) and Masini (137), arrived at very different results.

The former denied the existence of any differentiated representation of the larynx, or of other parts of the respiratory apparatus, in the cerebral cortex. Krause (138) has sufficiently answered this statement. Masini found in four cases, that *weak* stimulation of the phonatory cortical area produced at first isolated movements of the opposite vocal cord, followed by weaker movements of the vocal cord corresponding to the stimulated side. He is, so far as I know, the only experimenter who has obtained this result; in spite of the large number of controlling experiments concerning this point which Horsley and I have undertaken, both before and after the publication of Masini's statements, we have not been able once to corroborate them. Yet this is just the crucial point, whether *unilateral* cerebral lesions can produce organic unilateral laryngeal paralysis.

Although a certain number of cases have previously been published (139), (Andral, Gerhardt, Friedreich, Foville, Duval, Löri, Lewin, Livio Ronci, Luys, Cartaz, Massei, &c. (140), in which vocal disturbances or even laryngeal paralyses have been observed simultaneously with organic lesions of the higher centres, they cannot be utilised towards deciding the present question because they are all, in some way or other, incomplete. In some the diagnosis of a cerebral lesion is doubtful; in others multiple lesions exist; in a third category no laryngoscopic examination has been made; in others, again, either no autopsy was made or was described in a defective manner with regard to the condition of the medulla, the nerve fibres and muscles themselves.

In 1884 Bryson Delavan (141), called attention to what he considered as a possibility of localising, from clinical observation, the laryngeal cortical centre. Of the two cases, however, upon which he bases his views, in one (Seguin) no laryngoscopic

examination had been made; and he himself confessed (142) some years afterwards, that in his second case the laryngeal paralysis, supposed to be due to a cortical lesion, was of bulbar origin, as shown by the *post-mortem* examination.

Rosbach (143) has recently published an interesting paper, in which, from a carefully observed case, he draws the conclusion that unilateral paralysis of a vocal cord may be caused by lesion of the opposite insula. This is, however, invalidated by the considerations that there was atrophy of the corresponding side of the tongue, due to an undoubted bulbar lesion of the hypoglossal nucleus; that the integrity of the vocal accessory bulbar nucleus was merely *surmised* from the fact that the fibres of the nerve itself and of the laryngeal muscles had undergone no atrophy; and that he did not prove that the laryngeal paralysis had existed so long that atrophy must necessarily have followed.

In a recent paper Scheff (144) throws no light upon the pathogenesis of his cases; but the controversy between Messrs. Garel and Dor on the one hand, and Professor Horsley and myself on the other, which followed (145) the publication of a case of the French authors just named (146), is of importance for us.

Garel had previously reported (147) a case, from which he concluded that a unilateral laryngeal paralysis may be caused by cortical lesion of the opposite hemisphere. Horsley and I (148) at the time took exception to this view on the basis of our experiments.

Garel, in his paper with Dor, maintains his original view, and on the strength of the autopsy expands it into the statement that a unilateral lesion of the internal capsule also can produce *complete* paralysis of the vocal cord of the opposite side. To this interpretation Horsley and I made the following objections:—
1. In this case, according to the authors' own report, the medulla has not been *microscopically* examined, and therefore the absence of a lesion of this part is absolutely unproved. 2. The *complete* paralysis of the vocal cord (according to the report of the authors it was fixed in the "cadaveric position") can in no way be explained by a *cortical* lesion.

Physiological and pathological experience has shown that in the cortex of man and of the higher mammals so far examined (except the cat), not the *respiratory* but only the *phonatory* function of the larynx is specially represented. Thus, even if according to Masini's view, an isolated cross-effect could be exercised from one *phonatory* centre, the *vocal* but not the *respiratory* function of the larynx would suffer, the latter being

mainly, if not exclusively, governed by the *medulla*. The *absolute* immobility of the vocal cord in Garel and Dor's case, therefore, cannot possibly be traced up to a cortical lesion. Our view was not merely theoretical, for with sufficient caution one can extirpate *the entire cerebrum, including the internal capsule* down to the floor of the fourth ventricle, without interference, after the first shock, with the *respiratory movements of the vocal cords*.

The reply of the French authors to these objections was unsatisfactory. They declared: 1. That the microscopic examination of the medulla in their case had been unnecessary ("nullement necessaire.") 2. That they had spoken of cerebral only but not of medullary conditions.

In our final reply we left the decision as to the former point to the readers of the controversy, and concerning the second laid stress upon the fact that in this question the cerebral conditions can not be discussed without ever keeping in sight the medullary centres and the different laryngeal functions. From this point the principle involved lends importance to the controversy; and in all future cases of unilateral paralysis of a vocal cord, believed to be due to a lesion of the cerebrum on the opposite side, we must insist upon a *thorough microscopical investigation of the whole neuro-muscular apparatus involved, from the medulla downwards*, so as to exclude the possibility of a lesion below the higher centres.

I have here to make a few general observations concerning the relationship between clinical and pathologico-anatomical observation, and experiments on animals, so far as they bear upon the different conditions obtaining in paralysis of the abductors and in central paralyses. For it may be objected to my arguments that it is inconsistent, after objecting to the validity of experimental evidence in the question of abductor paralysis, to admit it in my criticisms of clinical and anatomo-pathological data concerning paralyses of cerebral origin. But this objection can tell only upon those who are personally unacquainted with experimental work. It is necessary, therefore, that purely clinical observers should realize that there exists in these two questions an enormous difference in the relative value of experimental investigation.

When dealing with cortical paralyses of the larynx, we stand on the very ground of experimental investigation. The localisation of special cortical areas for the motor innervation of the larynx, must be defined by experiment, just as experiment has

defined the localisation of other cortical areas. The physiological discoveries so made have uniformly been corroborated by pathological experience. The larynx forms no exception to this rule; and experimentation has added to our knowledge concerning its functions, without clashing with the teachings of clinical observation.

From the moment the question was raised as to the existence in *man* of a special bilateral cortical area for voluntary phonation, I have insisted upon the fact that even in typical cases of right sided hemiplegia with motor aphasia, there is no *aphonia*. In such cases no motor disturbances of the vocal cords can be seen; hence in man also there must exist a *bilateral* representation of the organs of phonation. Here therefore experiment and pathological experience agree. Now the seat of the *left* phonatory area corresponds to the seat of the *speech* centre, as shown to be highly probable by Horsley and myself in our investigations on monkeys; hence, if a unilateral cortical lesion could produce paralysis of the opposite vocal cord, it would follow that palsy of the right vocal cord and some phonatory trouble must occur in lesions of this area, nearly *as frequently and regularly* as aphasia and right-sided hemiplegia. But how few cases of this character have as yet been reported, though attention has already been long drawn to this point! Again, most of the cases thus reported are founded upon clinical observation only during life, the possibility of multiple lesions at a lower level being not excluded; whilst the records of few autopsies that have been made, are as already mentioned all deficient on some point or other.

But experiment teaches us even more. It shows us, what could indeed have been expected, that great differences exist in the central representation of the larynx, with regard to its *phonatory*, and to its *respiratory* function. It teaches us, that the former is mainly represented in the cortex, the latter in the medulla oblongata. It has even shown us how in a highly organised animal we can remove not only one or both phonatory centres, not only an entire cerebral hemisphere, but the *entire cerebrum* down to the floor of the fourth ventricle, without the *respiratory* movements of the vocal cords being altered in their character. *Such* experiments entitle us, when confronted with a case in which on the strength of a more or less incomplete clinical or pathological examination, a unilateral cortical lesion is said to have produced complete paralysis of the opposite vocal cord, to be sceptical, and suspect some fallacy of observation.

The case, however, is very different if, as in the question of

abductor paralysis, experiments are merely intended to *imitate a pathological process*, usually of a *chronic* nature. That imitations hardly ever come up to the original is as true in medicine as it is in other sciences. But if *nature itself* offers us experiments under conditions the diversity of which no experiment on animals can imitate, and yet with always the same results, we may well disregard any attempted imitation of pathological processes bearing a merely superficial likeness to its natural prototype and disallow the validity of any generalisation derived from such a delusive instance.

I am gratified here to find myself in agreement with no less an authority than Prof. Virchow. He, who in 1881 (149) had so warmly and eloquently defended experimental methods against prejudice, found himself three years later obliged to protest against *over-estimating* them in questions like the present. He was speaking of the ossification of the bone marrow at the border of an amputation wound, a process in which it is "the violence of the changes produced by the experiment" which distinguishes experimental from pathological phenomena. "Those observers," he says, in his discourse on metaplasia (150), "who demand that every organic process must first be demonstrated by vivisection deceive themselves, as it appears to me, about the importance of the experiment in such cases. Pathological conditions occur which may be observed with the same regularity as can be secured by experimental ways; nay, which are even preferable to experiment. . . ."

In further illustration of this thesis I may be allowed to give the outline of a case which I propose to publish fully on a future occasion.

Some years ago a woman, aged about 48, came to the Throat Department of St. Thomas's Hospital. Her only complaint was difficulty of swallowing, and she stated that she felt an obstacle about the level of top of the sternum. The patient looked sallow and cachectic. A bougie easily passed down the œsophagus. Though the symptoms did not point to any laryngeal affection, the left vocal cord was found to be fixed immovably in the *median position*, otherwise the larynx, as well as all other organs, were normal. The patient's age, the apparently normal state of all other organs, the absence of a syphilitic history, led one to think of an infiltrating carcinoma of the œsophagus, which had not yet obstructed the canal itself, but had already to some extent involved the left recurrent laryngeal nerve. Iodide of potassium was prescribed without any effect. A fortnight later there occurred ptosis of the left eyelid, then paralysis of the left rectus externus, followed shortly afterwards

by complete left ophthalmoplegia. Sensory troubles in the mucous membrane of the left cheek, left sided deafness, left sided paralysis of the facial nerve, made successively their appearance (154).

By this time of course we knew we had to deal not with a peripheral but with a central lesion on the left side, gradually involving one cerebral nerve after another. The patient was now admitted into the hospital, and subjected, under the care of Dr. Bristowe, to an energetic treatment by inunction, and large doses of iodide of potassium. Her condition within the next few months improved so much that, though not cured, she could leave the hospital. The left vocal cord remained all along immobile in the median position. Syphilitic pachymeningitis was diagnosed at the time. A few months later the patient, having become much worse, came back to the hospital; the paralyzes had returned with greater intensity; the left vocal cord was now fixed in the *cadaveric* position, and the voice, which had been formerly quite normal, had become almost aphonic. Signs of incipient paralysis were found in some of the right cranial nerves. The patient was submitted to the same treatment, but without success. The paralytic phenomena on the right side progressed, and after a while included *diminished outward mobility of the right vocal cord*. This paresis steadily increased, but before the stage of complete right abductor-paralysis had been reached the patient died comatose.

The autopsy corroborated the diagnosis of syphilitic pachymeningitis.

All the affected cranial nerves were found to be embedded, near their points of emergence from the cranium, in a half-gelatinous, half-fibroid mass, which had partly destroyed, partly compressed them. The vago-accessory nerves on both sides were involved.

The importance of this case is obvious. Nature had showed herself a perfect experimenter. This *one instance* supplies a clue to some of the most difficult problems which have arisen in the course of the controversy about abductor paralysis and contracture: Here is to begin with the transition of an originally observed left-sided abductor paralysis into *complete recurrent* paralysis. Here we have, as if to exclude the idea of a contracture primarily of the laryngeal muscles gradually passing into paralysis, *multiple* affections in other cranial nerve regions, all of which, though affected by the *same* process, showed only *paralytic*, and no irritative, phenomena. And if the objection were still raised that the proof was not complete, and that the original median position of the left vocal cord might possibly be due to some *irritation*, Nature enabled us to dispose of this doubt by allowing us to witness a *gradual diminution of the outward*

mobility of the right vocal cord, which cannot possibly be interpreted as an irritative phenomenon!

I hope I may not be misunderstood, when I ask what experiment could have faithfully reproduced the conditions of such a case?

Many years' clinical and experimental investigation leave no doubt in my mind that in these questions experiment cannot lay down the law to clinical observation, nor clinical observation to experiment. I feel equally certain there is no incompatibility between them, that the one *completes the other*, and that wherever a contradiction seems to arise the conflict is only *apparent*. With sufficient patience and care it can be always traced to defective clinical or pathologico-anatomical observation, to faults in the experimental method or to an incorrect way of stating the problem.

The importance of the questions just discussed which are of general as much as of special interest, has to some extent dwarfed the other problems of laryngeal nerve physiology and pathology, which have arisen since the introduction of the laryngoscope. Quite a number of important additions to our knowledge, however, are to be enumerated.

Thus the old question as to the primary source of the motor innervation of the larynx has recently attained a new phase through the papers of Grossmann (152) and Grabower (153). According to the former the superior laryngeal nerve in rabbits takes its origin from the upper, the recurrent laryngeal from the middle bundle of the vagus. Grabower's experiments on animals also led him to consider the vagus and not the accessory as the motor nerve of the larynx. But Koch (154) on anatomical grounds supports the generally accepted view that the spinal accessory is the motor nerve of the larynx.

Hints (155), in experiments undertaken by the degeneration method on rabbits, finds that the spinal accessory receives, in addition to those, from the motor and spinal roots, fibres from the jugular ganglion of the vagus, and that the recurrent laryngeal consists mainly of accessory fibres. Dees (156) on the other hand shares Grabower's view that the accessory is not the motor nerve for the larynx.

This problem, which after Schech's paper (*l.c.*) appeared to have been definitely solved in favour of the view that the spinal accessory is the only motor nerve for the laryngeal muscles proper,

except the crico-thyroid, is thus again in anything but a settled condition.

Without committing myself to a final opinion, I may mention that after section performed by Professor Horsley of the internal branch of the spinal accessory in a dog, I have seen the vocal cord of the same side at once recede into the cadaveric position, whilst the autopsy showed the *absolute integrity* of the corresponding vagus; and thus the results of Grabower and others are at present not intelligible to me. I shall return to this point when discussing cases of unilateral paralysis of the tongue, palate and larynx.

The innervation and the mode of action of the crico-thyroid muscle, is now also the subject of much controversy. Led by careful experiments, Hooper (157) arrived at the same conclusion as Magendie, that contrarily to the accepted opinion, the contraction of this muscle moves the cricoid cartilage towards the thyroid. This view, which had already previously been expressed by Jelenffy and Schech (*l.c.*), is shared by Moura-Bourouillou (158), Desvernine (159), Kiesselbach (160), and v. Beregszaszy (161).

The papers by Simanowsky (162), Exner (163), Onodi (164), and Breisacher (165), which discuss the innervation of the crico-thyroid and other laryngeal muscles are so contradictory between themselves that it is not possible as yet to form a definite opinion amidst such discrepancies.

The observations of paralysis of the crico-thyroid made by Heymann (166), and Kiesselbach (167) ought to be mentioned here; as well as the experiments concerning movements of the vocal cords produced by direct stimulation of the motor laryngeal nerves in man. Such experiments have been made by Gerhardt (168), von Ziemssen (169), Rossbach (170), Pauly (171), and Kaplan (172). But these experiments have also yielded such different results that further investigations are necessary before such data can be utilized.

The statement made by Hale White (173), that the recurrent exercises a trophic function upon the thyroid gland, and contains many fibres not of cerebro-spinal origin, has not so far received corroboration.

According to Moura (174) the inter-arytænoid muscle acts in concert with the glottis-openers, and Jacobson (175) ascribes the same function to the thyreo-arytænoid muscle.

The interesting experiment made by Fraenkel and Gad (176) by applying cold to the recurrent laryngeal, when the outward

movements of the vocal cord, innervated by the frozen nerve, cease sooner than the adductor movements, has already been mentioned, when the question of abductor paralysis was discussed. Onodi's (177) more recent papers, in corroboration of the law of the greater vulnerability of the abductors, are as yet mere preliminary communications, and hardly prove his thesis.

We now come to pathology. With regard to the central affections of the motor laryngeal nerves, we must first mention the corresponding chapter in Gottstein's (178) text-book of diseases of the larynx, which adduces an excellent collection of illustrative cases. Krause (179), Löri (180), and Neumann (181), have also made valuable contributions towards the etiology and symptomatology of laryngeal affections in central nervous disorders.

With regard to affections of the cerebrum proper, Arthur Schnitzler (182), Hirt (183), Scheinmann (184), and Michael (185), considering that the so-called functional aphonia is in the light of recent physiological investigations a *cortical* affection have treated it by hypnotism and suggestion.

A paper has been devoted by Rosenbach (186) to "Functional Paralysis of Articulate Phonation," under which term he includes cases in which there is apparent absence of all innervation as well as those in which only quantitative changes of innervation are present. B. Fraenkel (187), and Bresgen (188), describe as "Mogiphonia," the professional weakness or premature fatigue of the voice, an affection which, from the description of its authors, may as well belong to the class of central as of peripheral neuroses. A similar remark applies to the "inability of whispering" or "apsithyria," an affection described by J. Solis-Cohen (189), and Peltsohn (190). The interesting cases reported by Cartaz (191) have already been mentioned in connection with the question of abductor paralysis. In these, as well as in Broadbent's case (192), lesions were present, situated, in my opinion, *above* the medulla oblongata, though they are described by their authors as belonging to the domain of bulbar paralysis. My view would well explain the initial paresis of the *adductors* in voluntary phonation.

Among *bulbar* affections the first place is held by the laryngeal paralyses of *tabes dorsalis*. It is remarkable that, whilst motor paralyses of the larynx have met since 1860 with such general attention, the literature of tabid laryngeal paralysis, which within the last few years has enormously increased, dates

only from the year 1881. It is true that Féréol, Martin, Jean, Boudin, Krishaber, and others had previously drawn attention to the so-called "laryngeal crises" or attacks of spasmodic cough, which in more severe cases pass into laryngeal spasms, choking attacks, convulsions, and loss of consciousness. But it was only in 1881 that Cherchevsky (193), who collected older cases and added some new ones from Charcot's clinic, stated that in some of these cases there had been actual laryngeal paralysis, and thus opened this new pathological chapter.

The neglect which has so long attended these paralyses, though strange, admits however of a very natural explanation. In a recent monograph on "The Laryngeal Troubles of *Tabes Dorsalis*," by Dr. H. Burger, (Leyden, E. I. Brill, 1891), it is shown that in the great majority of cases of *tabes*, in which laryngeal paralyses have been observed, the paralysis met with is of the nature of *abductor* paralysis. This, unless bilateral and fully developed, easily escapes observation, as mentioned by Schech in 1871, and emphasized by myself ten years afterwards, because *it leaves the voice wholly unimpaired*, whilst the unilateral form does not cause any *respiratory* symptoms, and the bilateral only, when much developed. And just in cases of *tabes*, in which the patients often are confined to bed, and do not exert themselves, even more advanced stages of bilateral abductor-paralysis often cause no appreciable dyspnoea. It is only since we have learned to appreciate the *diagnostic* value of this form of paralysis, and begun to *look* for it in suspicious cases, as we do for optic neuritis, that the number of cases of *tabes* in which laryngeal paralyses have been observed, has increased. We shall allude here only to a few of the contributions, the complete literature of the subject being given in Burger's paper.

My first case of bilateral abductor paralysis in *tabes* having apparently fallen into oblivion, I will mention it here because it is the *first* one of bilateral abductor paralysis in *tabes*, which has been authenticated by laryngoscopic examination, and because it is the very case which gave rise to the development of the law of the greater vulnerability of the abductors in progressive organic nerve-lesions. It is my own fault that it has been generally overlooked, because I did not realise at the time (1878) the connection between the laryngeal symptoms, then of two years' date, and the other incipient *tabid* symptoms. I only later arrived at a correct understanding of its etiological conditions. The reader who will take the trouble of following up the case in the different writings in which I have referred to it (194), or look it up in Burger's mono-

graph (*l.c.* p. 9), where it is fully given, may be compensated for his trouble by the exceptional interest it presents. One of its most remarkable features is the circumstance that in it, as in the well-known case observed later by Weil (195), the laryngeal symptoms preceded the usual symptoms of tabes by *fully two years*.

Important papers concerning paralytic laryngeal symptoms in tabes have also been contributed by M'Bride (196), Munschina (197), Oppenheim (198), Landgraf (199), Ross (200), Saundby (201), Kraus (202), Martius (203), Küssner (204), Felici (205), Aronsohn (206), Lucas Championnière (207), Dreyfuss (208), van Gieson (209), and Fano-Marina (210).

The two problems now before us, and which, I think, ought to be solved in the near future are:—(1) The relative frequency of these symptoms; and (2) the mutual relationship between crises and paralyses.

Concerning the first point apparently irreconcilable statements have been made by different authors. The explanation is, in my opinion, to be found in the fact that these statements are mostly based upon *too small* a material, and my own experience concerning this point is instructive enough. When I first began to study this question from the clinical point of view in the National Hospital for Paralysis and Epilepsy, I found among the first twelve cases of tabes which I examined not less than seven, in which either unilateral abductor paralysis, unilateral recurrent paralysis, or bilateral advanced paresis of the abductors, was present. I was surprised at this enormous proportion, and could hardly understand how, in these circumstances, the participation of the larynx in tabes should so long have escaped notice. On continuing, however, my observations, by methodical laryngoscopy of *every* case of tabes which came into the Hospital, I did not find among the next fifty to sixty patients *a single case* with paralytic laryngeal symptoms! In a more recent series of observations extending over fifteen months, and comprising all the in-patients suffering from tabes admitted into the Queen Square Hospital, I found in 27 cases of tabes seven instances of unilateral or bilateral paralysis or paresis of the glottis-openers. It is easy to understand from these statements how it happens that the reports on the frequency of laryngeal paralysis in tabes by authors who have worked on a comparatively small material only should be so widely discrepant.

Concerning the mutual relations of crises and paralysis no necessarily causal connection according to my experience, exists

between them. In a number of cases paralyzes (unilateral or bilateral paresis or paralysis of the abductors or complete recurrent paralysis) are met with without any previous crises; in a second series, no paralysis ensues, even after occurrence of frequent and severe crises; whilst in a third series both, spastic and paralytic, phenomena are met with together in the same case. Should paralyzes be produced, the law of the greater vulnerability of the abductors holds good. The spastic phenomena must be traced, I think to an increased irritability of the adductor centres. A peripheral stimulus conducted along the centripetal fibres of the superior laryngeal to those centres which are, according to this theory, in a condition of increased irritability, does not set up a mere cough, as under normal conditions, but spasmodic coughing, spasm of the glottis, general convulsions, in short a "crisis." This view also explains naturally the otherwise obscure influence of applications of cocaine upon the larynx, the benefit of which is mentioned by several authors (Gerhardt, Landgraf, Krause, &c.), the peripheral stimuli being temporarily cut off from the excitable centres. I shall conclude these remarks with a translation of the conclusions of Burger in his excellent monograph.

1. "The medulla oblongata is very frequently involved in the pathological process of *tabes dorsalis*.

2. Among bulbar nerves the vago-accessory is by far the most frequently attacked.

3. The morbid phenomena observed in the laryngeal nerves in *tabes* are motor paralyzes, troubles of co-ordination, and so-called laryngeal crises.

4. The motor paralyzes are the commonest form of laryngeal lesion; they often occur, however, without producing any symptoms.

5. The literature of tabid laryngeal paralyzes furnishes a complete corroboration to Semon's law. Abductor paralysis is the tabid laryngeal paralysis "par excellence." No authenticated reports exist of isolated adductor paralyzes.

6. The course of these paralyzes is usually slow and progressive; the prognosis is always unfavourable.

7. In progressive tabid laryngeal paralysis, next to the posticus, the internal thyreo-arytænoid muscle is the adductor first affected.

8. Transitory functional adductor paralyzes occurs occasionally in the progress of *tabes*.

9. Paralyzes in the domain of the superior laryngeal are rare

in tabes, and have been observed only in connection with paralysis in the region of the inferior laryngeal nerve.

10. Other paralytic phenomena in the territory of the accessory nerve are pretty often associated with the laryngeal paralysis.

11. The tabid fixation of the vocal cords in the median line is a consequence of secondary contracture of the adductors, which has been superadded to a primary paralysis of the posticus.

12. Ataxy of the vocal cords exists.

13. In normal abducting and adducting movements both the antagonistic groups of muscles of the larynx are probably innervated. Both physiological analogy (movements of the limbs) and pathological phenomena (*motus perversus* in respiration in cases of abductor paralysis and the tabic ataxy of the vocal cords) support this view.

[I have taken exception to this view in my paper on the position of the vocal cords in quiet respiration already quoted, and in my review of Burger's Monograph *Internationales Centralblatt für Laryngologie, &c.*, 1892, p. 354 *et seq.*]

14. Under the collective name of "laryngeal crises all attacks of dyspnoëic cough observed in tabes have been put together. These are of a threefold nature: (a) Attacks which are due to defective closure, or anæsthesia of the aditus ad laryngem. (b) The laryngo-spastic attacks of suffocation which accompany abductor paralysis. (c) The laryngeal crises in the strict sense.

15. The laryngeal crises proper (c) occur either alone, or connected with laryngeal paralysis.

16. They are due to a condition of irritability in the sensory tracts of the laryngeal nerves, usually connected with an increased reflex excitability of the adductor centres.

17. The irritation which produces the attacks depends upon a chronic degeneration of the nerve tissue, and is usually localised in the *medulla oblongata*.

18. The anatomical basis of tabid laryngeal paralysis consists in a degeneration of the vago-accessory nuclei or fibres in the medulla, coupled with degenerative atrophy of the vagus and recurrentes, and of the paralysed muscles (*Mm. crico-arytenoidei postici*)."

Such are Burger's conclusions. This is not the place to enter more fully upon the interesting questions raised, and which form a fruitful field for further research.

Among other medullary affections we may mention the interesting association of unilateral paralysis of the tongue, palate and larynx, first described by Hughlings Jackson (211), and after

him by Stephen Mackenzie (212), Barlow (213) (no laryngoscopic examination), Oltuszewski (214), Limbeck (215), Turner (216) and (?) Jonathan Wright (217). The main interest of these cases was considered to lie in the light which this curious association throws upon the innervation of the *soft palate*, inasmuch as it implies that the motor innervation of this organ is supplied by the accessory, in agreement with the experimental results of Horsley and Beevor (218), and of Horsley and myself (219). These cases gain still further interest from Grabower's assertion (see above) that the pneumogastric, not the accessory, supplies motor innervation to the larynx. This assertion is the less supported by these cases, that in several the cucullaris and trapezius were also paralysed, and thus *both* branches of the accessory were affected, whilst there was no symptom pointing to an affection of the vagus.

Among other papers concerning laryngeal bulbar paralyses, I need only mention some well described cases by Eisenlohr (220); and Remak's (221) case of bilateral paralysis of the spinal accessory nerves. Not much can be made, I think, of Lenmalm's (222) case of paralysis of the vocal cords in amyotrophic lateral sclerosis.

Finally we come to *peripheral* affections, concerning which we find papers by Jankowski (223) and Rotter (224) (laryngeal paralysis after extirpation of goitre), Bäumlér (225) (paralysis of the recurrent laryngeal nerve in chronic affections of the lungs), Newman (226) (laryngeal symptoms due to pressure of aneurisms upon the vagus and the recurrent laryngeal nerves), Möser (227) (laryngoscopic image in complete unilateral paralysis of the vagus nerve).

Comparatively little attention has been paid to the paralyses of the muscles antagonistic to the abductors, if we except the central cases, with functional paralysis of the entire adductor apparatus. Still, here also a number of interesting papers may be recorded. Thus Frank Donaldson, jun. (228), reports a case of paralysis of the lateral adductor, and W. R. H. Stewart (229) two cases of unilateral adductor paralysis due to reflex influences from the nose. French laryngologists have thoroughly studied the genesis, symptomatology and treatment of paralysis of the transverse arytaenoid muscle, the papers of Lecointre (230), Lermoyez (231), and Proust and Tissier (232) deserving special mention.

I am at the end of my description, which, let me repeat, is not intended to refer to *every* publication made concerning the question of motor laryngeal paralyses, in the course of the last thirty years, but only to those which, according to my conviction, have contributed some solid elements to our knowledge. I have hardly alluded to Sensory paralyses and Spasmodic affections of the larynx.

Nevertheless, I venture to believe that those readers who have not paid special attention to the questions treated in this paper will, after reading it, be surprised to discover so great an activity in this apparently remote corner of medical investigation, and to find how the questions which move scientific laryngology overlap other domains of pathology. More has been done within the last thirty years on these topics alone than during the whole pre-laryngoscopic period. But few of the questions raised, however, have been definitely settled, and on almost all the points to which I have had to refer, important problems still await elucidation. And, again, many points will become the objects of investigation in the near future, of which we have hitherto hardly had a glimpse.

I conclude with expressing the wish that in the further development of the subject we may be spared any further attempt at founding a special laryngeal pathology; and that it may be always remembered that the true scientific course is to refrain from premature explanations of observed facts. The true causation must eventually be reached by quietly and patiently following up and carefully comparing observations. He who is led away by mere ingenuous views, like the man who follows a Will-o'-the-Wisp, loses time and often lands in a morass.

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- (5) *Berliner klinische Wochenschrift*, 1869, p. 383.
- (6) *New York Medical Journal*, Nov. 1880, p. 460.
- (7) Vgl. z. B. "Ueber Diagnose und Behandlung der Stimmbandlähmung," *Volkmann's Sammlung klinischer Vorträge*, No. 36, 1872. "Laryngologische Beiträge." *Deutsches Archiv für klin. Medicin*, 1873, Bd. 11, p. 575. "Stimm-bandlähmung und Icterus," *Deutsche med. Woch.* No. 10, 1887, etc., etc.
- (8) "Ueber Stimmbandlähmung." *Wiener medicinische Presse*, 1866, No. 417, ff.

- (9) "Ueber Stimmbandlähmung." *Deutsches Archiv. für klin. Med.*, No. 2, p. 515, 1867, und *Transactions of the Pathological Society of London*, vol. 23, p. 66.
- (10) *Trans. Path. Soc.*, vol. 24, p. 42.
- (11) "Stimmbandlähmungen." *Deutsches Archiv. für klin. Med.*, Bd. 4, p. 376.
- (12) "Cases of Laryngeal Paralysis." *Medical Times and Gazette*, 3. April, 1869.
- (13) "Ueber Recurrenslähmungen." *Deutsches Archiv. für klin. Medicin*, Bd. 6, p. 37, 1869.
- (14) "Cases of Aphonia from Paralysis of Intrinsic Muscles of the Larynx." *Amer. Journ. of Med. Science*, p. 305, 1870.
- (15) "Diagnosticque de la paralysie motrice des muscles du larynx." Paris, 1872.
- (16) *Edinburgh Medical Journal*. July, 1881.
- (17) *Transactions of the Pathol. Society*. 1882, vol. xxxiii., p. 82.
- (18) *Sperimentale*. März, 1884.
- (19) *Wiener Med. Presse*. No. 20, 1882.
- (20) *Berliner klin. Wochenschrift*. No. 50, 1882.
- (21) *Ibidem*. No. 1, 1883.
- (22) *Klinik der Krankheiten des Kehlkopfes*. Wien, 1866.
- (23) "Hoarseness, Loss of Voice and Stridulous Breathing in relation to Nerve and Muscle Affections of the Larynx." London, Churchill & Sons, 1868.
- (24) "Diseases of the Throat and Nasal Passages." Second edition, Philadelphia, 1879.
- (25) "Ueber die Functionen der Nerven und Muskeln des Kehlkopfs." *Habilitationschrift*. Würzburg, 1873.
- (26) "Laryngoscopie an Thieren." *Berliner Wochenschrift*, 3. October, 1873.
- (27) *Medical Times & Gazette*, p. 638, 1866.
- (28) "Hoarseness and Loss of Voice," etc., p. 34.
- (29) *Berliner klinische Wochenschrift*, 1872, No. 20 und 21 und 1870, No. 7.
- (30) *Volkmann's Sammlung klinischer Vorträge*. No. 95.
- (31) *Ueber die Paralyse der Glottiserweiterer*. *Deutsches Arch. für klin. Med.* Bd. 13, 1874, p. 107 ff.
- (32) *Loc. cit.*, p. 462 ff.
- (33) "Paralyse der Musculi crico-arytaenoidei postic." *Berliner klin. Wochenschrift*, 1879, No. 33.
- (34) *Loc. cit.*, p. 460.
- (35) *Transactions of the Clinical Society of London*, vol. xi., p. 141 ff.
- (36) *Loc. cit.*, p. 453.
- (37) *Berliner klin. Wochenschrift*, 1883, No. 46.
- (38) "Diseases of the Throat and Nose," vol. i., 1880.
- (39) *Breslauer ärztliche Wochenschrift*, No. 2 u. 3, 1880.
- (40) *Berliner klinische Wochenschrift*, No. 17 u. 22, 1884, No. 8 u. 10, 1888.
- (41) Vol. ii., No. 3, Juli, 1881.
- (42) *Loc. cit.*, p. 205.
- (43) *Loc. cit.*, p. 220.
- (44) *Loc. cit.*, p. 594.
- (45) The reports of cases relating to this question and published during the first few years after the publication of my paper, have been fully quoted in my paper, "Ueber die Lähmung der einzelnen Fasergattungen des Nervus Laryngeus inferior (recurrens), *Berliner klin. Wochenschrift*, 1883, p. 46, *et seq.* A contribution by the late Professor Elsberg (*Philadelphia Medical Times*, July 30, 1881) is important. It shows that as the abductor fibres are more vulnerable than the abductors to organic pathological influences attacking the whole nerve trunk, so they recover more slowly and rarely from such lesions. I have lately had the opportunity of repeatedly corroborating this observation (see for instance *British Medical Journal*, Nov. 30, 1889, Meeting of the Medical Society of London).
- (46) *Transactions of the International Medical Congress*, London, 1881, vol. iii., p. 214 ff.

- (47) *Loc. cit.* p. 218.
- (48) "On the Position of the Vocal Cords in Quiet Respiration in Man, and on the Reflex Tonus of their Abductor Muscles," *Proc. Royal Soc.*, vol. xlviii., 1890.
- (49) *Comptes Rendus du 8 Congrès International*, vol. iv., p. 43 ff.
- (50) *Berliner klinische Wochenschrift*, 1883, No. 46 ff.
- (51) How negligently some authors have proceeded here appears, e.g., from the fact that in an otherwise excellent recent text-book on "Affections of the Larynx," by J. E. Moure ("Leçons sur les Maladies du Larynx," 1890, p. 313 *et seq.*) the author represents me as the *originator* of the different hypotheses, which I have *contested* in my first paper, and devotes to me a long polemic!
- (52) *Berliner klin. Wochenschrift*, 1884, p. 138.
- (53) *Ibid.*, 1884, No. 14.
- (54) *Ibid.*, 1884, p. 734.
- (55) "Bericht über die Sommersaison 1884 im Schwefelbad Langenbrücken," Bruchsal, D. Weber. 1885.
- (56) "A Contribution to the Study of Laryngeal Paralysis," *Edinburgh Med. Journal*, Juli, 1885.
- (57) "Zwei Fälle von doppelseitiger Lähmung der Musc. crico-arytaenoidei postici," *St. Petersburger med. Wochenschrift*, No. 26-28, 1884.
- (58) "Ophthalmoplegia, etc." *BRAIN*, October, 1885.
- (59) "Aneurism of the Ascending and Transverse Aorta, etc." *British Med. Journal*, 19th December, 1885.
- (60) "A Case of Paralysis of the Abductors of the Vocal Cords, etc." *St. Bartholomew's Hospital Reports*, vol. xxii., 1886.
- (61) "Bilateral Paralysis of the Dilator Muscles of the Glottis with subsequent Paresis of the Constrictors." *Lancet*, 16 and 23 July, 1887.
- (62) "Paralysis of the Abductors of the Vocal Bands." *Lancet*, 22 and 29 October, 1887.
- (63) "Extirpation eines Cavernoms, etc." *Berl. klin. Woch.*, No. 7, 1888.
- "Traumatische Sympathicus-Hypoglossus- und Accessoriusparalyse," *Ibid.* ; "Bemerkungen zu Remak's Fall, etc." *ibidem*, No. 8.
- (64) "Zur Pathologie der Glottiserweiterung." *Deutsche med. Woch.* No. 26-28, 1888.
- (65) "Ueber Recurrenslähmungen." *Münchener med. Wochenschr.*, No. 51, 1888.
- (66) "Ein Fall von linksseitiger Stimmbandlähmung, etc." *Charité-Annalen* xiii., 1888.
- (67) "Zwei bemerkenswerthe Fälle von Stimmbandlähmung." *Ibid.*, xiv., 1889.
- (68) "Die Lähmungen der Kehlkopfmuskulatur im Verlaufe der Tabes dorsalis." *Virchow's Archiv*, 120, i., 1890.
- (69) "Klinische Beiträge zur Kenntniss der Innervation, etc., des Kehlkopfes." *Orvosi Hetilap*, No. 25 ff., 1890, und *Pest. Med.-chir Presse* No. 36, 1890.
- (70) *Berliner klin. Wochenschrift*, 1881, No. 50.
- (71) *Archivi italiani di Laryngologia*, 1882, p. 52, und *Bolletino della Malattia dell' Orecchio*, No. 2, 1886.
- (72) *BRAIN*, vol. v., p. 53, and "Diseases of the Nervous System," p. 199.
- (73) *BRAIN*, vol. vi., p. 266.
- (74) *Ibid.*, vol. iv., No. 14 and 15.
- (75) "Kehlkopfkrankheiten," *Erste Auflage*, 1884, Kapitel über Motilitätsneurosen.
- (76) "La Paralisis de los Musculos Crico-aritenoides post, etc." Madrid, 1884. T. Amparo.
- (77) "Etude sur la Contractilité Post-mortem, etc." *Arch. de physiol. normale et pathol.*, No. 6, 1885.
- (78) "On an apparently Peripheral and Differential Action of Ether upon the Laryngeal Muscles." *Brit. Med. Journal*, 28th August and 4th September, 1886.
- (79) "Roaring in Horses." London, Baillière, Tindall & Cox, 1889.
- (80) "Roaring in Horses." *Illustrated Naval and Military Magazine*, vol. ix., August, 1888.

(81) "Ueber doppelseitige Recurrenslähmung." *Wiener medizinische Presse*, No. 18, 1882.

(82) "Some points in relation to the diagnostic significance of immobility of one vocal band, etc." *American Journal of Medical Sciences*, July, 1883.

(83) "Paralysis of the left vocal band in extreme abduction." *Transactions of the American Laryngological Association*, 1884.

(84) "Les fibres abductrices des Recurrents sont-elles toujours affectées les premières?" *Revue Mensuelle de la Laryngologie*, No. 5, 1886.

(85) *Berliner klinische Wochenschrift*, 1883, No. 46 ff.

(86) *Deutsche med. Wochenschrift*, No. 27, 1885.

(87) "Note sur la paralysie laryngée d'origine centrale." *La France Médicale*, No. 134 et 135, Vol. ii., 1885.

(88) *Berliner klinische Wochenschrift*, No. 35, 1884. (In cases of undoubtedly bulbar lead-paralysis of the laryngeal muscles the law of the greater vulnerability of the abductors is exemplified. (See for instance, Remak, "Saturnine Hemiatrophie of the Tongue." *Berliner klinische Wochenschrift*, No. 25, 1886.)

(89) I have demonstrated specimens of this kind before the Pathological Society of London and before the Laryngological Section of the Berlin Naturforscherversammlung in 1886.

(90) "Quelques reflexions à propos de l'affection dite paralysie des dilatateurs de la glotte." *Annales des maladies de l'oreille, etc.* Janvier, 1886. "Paralysie des dilatateurs de la glotte." *Ibid.* Septembre, 1886, etc.

(91) "Sur la paralysie des muscles dilatateurs de la glotte." *Bull. de la Soc. des Sciences méd. du Grand-Duché de Luxembourg*, 1885.

(92) "Etude anat. et pathol. des ganglions peritrachéo-laryngiens." *Annales des mal. de l'oreille, etc.* März, 1884.

(93) "Diagnosis of Diseases of the Brain," 1885, p. 101.

(94) "Endemic Goitre or Thyrotoe." London, J. and A. Churchill, 1885.

(95) *Transactions of the Pathological Society of London*, 1882.

(96) "Innervatie. Functie en Verlaming van enkele Larynxspieren." Leiden, S. C. van Doesburgh, 1886.

(97) *Sitzungsbericht der k. k. Akademie der Wissenschaften in Wien*, vol. lxxxix. Abth. 3, 1. u. 2. Heft, 1884.

(98) "Studien über Innervation und Atrophie der Kehlkopfmuskeln." *Ibid.*, vol. lxxxv.

(99) "Zur Anatomie der Kehlkopfnerven." *Ibid.*, vol. lxxxv.

(100) See, e.g., a case from the second table of my paper in the *Berliner klinische Wochenschrift*, 1883; also the paper above quoted by Percy Kidd; and my own case, which will be published in full in this paper, etc.

(101) "Three Cases of Pressure upon the Recurrent Laryngeal Nerve." *Transactions of the College of Physicians of Philadelphia*, May 5, 1886.

(102) "Beiträge zur Anatomie, Physiologie und Pathologie der Kehlkopfmuskeln." *Berliner klin. Wochenschr.*, No. 34 u. 36 und *New York Med. Jour.*, Aug. 31st, 1889.

(103) "Contribution à l'étude des sténoses glottiques chez les tuberculeux." *Annales des mal. de l'oreille, etc.*, Januar, 1887.

(104) "Medianstellung des Stimmbandes bei Recurrenslähmung," &c. *Virchow's Archiv.*, vol. 120, 124, 126.

(105) "Ueber physiologische Verschiedenheiten der Skelettmuskeln." *Breslauer ärztliche Zeitschrift*, No. 18 u. 24, 1883 und "Zur Anatomie und Physiologie der quergestreiften Muskeln." *Recueil Zoologique Tome, I.*, No. 4, 1884.

(106) "Ein Versuch zur Erklärung des Unterschiedes im Verhalten der Glottisschliesser und -erweiterer bei Kehlkopflähmungen centralen Ursprungs" (in Russian), *Getchen klin. Gaz.*, No. 26 to 29, 1885. Referat im *Internationalen Centralblatt für Laryngologie, etc.*, Febr., 1886.

(107) "On an apparently peripheral and differential action of ether upon the laryngeal muscles." *B. M. J.*, 4 and 11 Sept., 1886.

(108) "The respiratory function of the human larynx." *New York Med. Journal*, July 4, 1885.

(109) "The function of the recurrent laryngeal nerve." *The Amer. Journ.*

of the *Med. Sciences*, July 1886, and *New York Med. Journal*, 6 and 13 Aug., 1887.

(110) *Brit. Med. Journ.*, 4 and 11 Sept., 1886.

(111) "Untersuchungen und Studien über Contracturen der Stimmbandmuskeln." *Virchow's Archiv.*, Bd. 98, Heft 2, p. 294.

(112) "Zur Aetiologie der Stimmbandlähmungen." *Deutsches Archiv für klin. Med.*, vol. 37, Heft 6, 1885.

(113) "Quelques reflections à propos de l'affection dite paralysie des dilateurs de la glotte." *Annales des maladies de l'Oreille et du Larynx*, No. 1, 1886.

(114) "Three cases of pressure upon the recurrent laryngeal nerve, etc." *Trans. of the College of Physicians of Philadelphia*, 1886.

(115) "Leçons sur les maladies du larynx." 1890, Paris, p. 313 ff.

(116) "Contributions à l'étude des névrites primaires du tronc du recurrent." *Revue Mensuelle de la laryngologie, etc.*, No. 20, 1889.

(117) *Verhandlungsberichte*, Jahrgang, 1883 u. 1884, No. 17 u. 18.

(118) *Comptes Rendus du Congrès.* Vol. iv., Copenhagen, 1886, p. 43 ff.

(119) "Ueber das Verhalten der Schliesser und Oeffner der Glottis bei Affectionen ihrer Nervenbahnen." *Virchow's Archiv.*, Bd. 99, Heft 1, 1885.

(120) *Internationales Centralblatt für Laryngologie, etc.* Oct., 1885, p. 87.

(121) a. "Ueber die Adductoren-Contractur (vulgo Posticuslähmung) der Stimmbänder." *Virchow's Archiv.*, Bd. 102, 1885.

b. "Zur Pathogenese der Adductoren-Contractur der Stimmbänder." *Deutsches Archiv für klin. Med.*, Bd. 38, Heft 5 u. 6, 1886.

(122) "Posticus-Lähmung oder Adductoren-Contractur." *Sitzungsberichte der Würzburger Physico-medizinischen Gesellschaft*, 1885.

(123) "Aphonia und Dyspnoea spastica." *Wiener med. Presse*, No. 41 u. 42, 1885.

(124) "Lähmung der Glottiserweiterer als initiales Symptom der Tabes dorsalis." *Berliner klin. Wochenschr.*, No. 13, 1886.

(125) *Tageblatt der 59. deutschen Naturforscherversammlung.* Berlin, 1886, p. 226.

(126) The excerpts from Krause's and Remak's observations in the discussion have been taken from a shorthand report which was made for the "Internationales Centralblatt für Laryngologie" of the Meeting of the Laryngological Section of the Naturforscherversammlung, 1886. This report is in my possession and will be published.

(127) "Ueber die Beziehung des Kehlkopfs zum motorischen Nervensystem," *Deutsche med. Wochenschrift*, 1890, No. 31.

(128) *Transactions of the American Laryngological Association*, vol. 7.

(129) *Brit. Med. Journal*, 4 and 11 Sept., 1886.

(130) "Versuche über die Ausfallserscheinungen der Stimmbandbewegung bei Abkühlung des Nervus recurrens." *Centralblatt für Physiologie*, 11 Mai, 1889.

(131) "The Functions of the Brain," 1876.

(132) "Traumatismes cérébraux," 1878, p. 1842.

(133) "Ueber die Beziehungen der Grosshirnrinde zu Kehlkopf und Rachen." *Sitzungsber. d. königl. preuss. Akad. d. Wissensch. zu Berlin*, Nov., 1883, und *Arch. f. Anat. u. Physiol. Phys. Abth.*, 1884.

(134) *Brit. Med. Journal*, 21 Dec., 1889; *Berliner klin. Wochenschrift*, 1890, No. 4. *Deutsche med. Wochenschrift*, 1890, No. 31. *Phil. Trans. of the Royal Society of London*, vol. clxxxii, B., 1890. This is not the place to enter more fully upon the unnecessary and unpleasant controversy which Krause began after the publication of our preliminary communication in the *British Medical Journal*, in defence of his priority, which we had admitted on four occasions in 1886 and 1887. This controversy will be found in the early numbers of the *Berliner klinische Wochenschrift* of 1890. I mentioned it here because the replies by Horsley and myself contain points of importance about laryngeal paralysis.

(135^a) *British Medical Journal*, 1890.

(135^b) "Note on Bilateral Degeneration in the Pyramidal Tracts, etc." *Brit. Med. Journal*, 4 January, 1890.

(136) "Leçons sur la fonction motrice du cerveau." Paris, 1887, p. 146 to 148.

(137) *Archivi italiani di Laryngologia*. Napoli, April, 1880, p. 45.

(138) "Zur Frage der Localisation des Kehlkopfs in der Grosshirnrinde." *Berl. klin. Wochenschrift*, No. 25, 1890.

(139) The following description is identical with that given by Horsley and myself in our paper "On the Central Motor Innervation of the Larynx" (*Transactions of the Royal Society*, 1890).

(140) The literature of these cases will be found in Masini's, Garel and Dor's, and Rossbach's papers, to be presently discussed. I do not refer to them individually, because the authors named themselves admit that none of them possess a decisive value for our purpose.

(141) *Comptes Rendus des travaux de la Section de la Laryngologie du huitième Congrès International périodique des Sciences médicales*. Copenhague, 1884, p. 70.

(142) *Transactions of the American Laryngological Association*, 1888, p. 195. Vgl. a. *New York Med. Journ.*, 22 June, 1889.

(143) "Localisation des corticales Stimmcentrums beim Menschen." *Deutsches Archiv für klin. Medizin*, Bd. 46, März, 1890.

(144) "Zur Pathologie der Motilitätsstörungen des Kehlkopfs, etc." *Allg. Wiener Med. Zeitung*, No. 22, 1890.

(145) *Annales des Maladies de l'oreille et du larynx*, April, 1890.

(146) *Ibid*, Mai und Juni, 1890.

(147) *Ibid*, Mai, 1886.

(148) *Lancet*, 1886.

(149) *Trans. of the 7th Med. Congress*, London. 1881, vol. i.

(150) Vortrag, gehalten auf dem Internationalen Medicinischen Congress in Kopenhagen. 1884, Separatabdruck, p. 14.

(151) I here report from memory, inasmuch as the notes of the case are at present in Dr. Bristowe's possession, and it is possible that the order of occurrence of the different paralyses may have been slightly different.

(152) *Wiener klin. Wochenschrift*, No. 49, 1889, No. 1, 3 u. 5, 1890.

(153) *Centralblatt für Physiologie*, No. 20, 1890.

(154) *Hospitals Tidende*, No. 20, 1890.

(155) *Pesther Med.-Chir. Presse*, No. 23, 1888.

(156) *Archiv. für Psychiatrie*, No. 20, 1 Oct., 1888.

(157) *Transactions of the American Laryngological Association*, 1883.

(158) *Comptes Rendus de la Société française d'Otologie et de Laryngologie*, 1884.

(159) *Journal of Laryngology*, Feb. 1888.

(160) *Monatsschr. für Ohrenheilkunde*, No. 3, 1889.

(161) *Arch. f. d. gesammte Physiologie*, 46, 10, 1890.

(162) *Getschen. klin. Gaz.*, No. 26, 1887, und *Pflüger's Archiv.*, Bd. 42, 1888.

(163) "Bemerkungen über die Innervation des Musculus Crico-thyreoideus." *Arch. f. d. gesammte Physiologie*, Bd. 43, 1, 1888. "Ein physiologisches Paradoxon, betreffend die Innervation des Kehlkopfs." *Centralblatt für Physiologie*, 6, 1889.

(164) "Beiträge zur Lehre von der Innervation und den Lähmungen des Kehlkopfs." *Monatsschrift für Ohrenheilkunde*, No. 4, 1888.

(165) "Versuche über den Nervus Laryngeus superior." *Centralblatt für die med. Wissenschaften*, No. 43, 1889.

(166) "Zwei Fälle von Lähmung des M. crico-thyreoideus." *Arch. f. klin. Med.*, vol. xlv., 5-6. Juni, 1889.

(167) "Zwei Fälle von Lähmung des M. crico-thyreoideus." *Arch. f. klin. Med.*, vol. xlv., 3-4. Oct., 1889.

(168) *Virchow's Archiv.*, 1863, vol. 27.

(169) "Die Electricität in der Medicin." Dritte Auflage, 1866, p. 198.

(170) *Monatsschrift für Ohrenheilkunde*, No. 10, 1881.

(171) Quoted by Kaplan.

(172) "Experimenteller Beitrag zur electrischen Erregbarkeit der Glottis-musculatur." *Inaug. Diss.*, Berlin, 1884.

(173) *Brit. Med. Journal*, 22 August and 28 Nov., 1885.

- (174) *Revue Mensuelle de la Laryngologie*, No. 12, October, 1886.
- (175) "Zur Lehre vom Bau und der Function des Musculus thyreo-arytae-noideus beim Menschen." *Arch. für microscopische Anatomie*, Bd. xxix, 1887.
- (176) *Centralblatt für Physiologie*, 11 Mai, 1889.
- (177) "Eine neue experimentelle Methode im Gebiet der Kehlkopfmuskeln und Nerven." *Berliner klinische Wochenschrift*, No. 18, 1889, und *Pester Med.-Chir. Presse*, No. 16, 1889. "Experimentelle Untersuchungen zur Lehre von den Kehlkopflähmungen." *Centralblatt für die med. Wissenschaften*, No. 15 u. 16, 1889. "Zur Lehre von den Kehlkopflähmungen." *Ibid.*, No. 40, 1889.
- (178) "Die Krankheiten des Kehlkopfs." 3. Aufl., 1890, Leipzig u. Wien, F. Deuticke, p. 344 ff.
- (179) "Ueber Functionstörungen des Kehlkopfs, etc." *Centralblatt für Nervenheilkunde*, No. 23, 1885.
- (180) "Die durch anderweitige Erkrankungen bedingten Veränderungen des Rachens, des Kehlkopfs und der Luftröhre." Stuttgart, 1885, F. Enke.
- (181) "Lecture on Aphonia, etc." *Glasgow Med. Journal*, July—October, 1885.
- (182) "Ueber functionelle Aphonie, etc." *Internat. klin. Rundschau*, No. 10-14, 1889.
- (183) "Ueber die Bedeutung der Suggestionstherapie, etc." *Wiener Med. Woch.*, No. 27-30, 1890.
- (184) "Die suggestive Tonmethode, etc." *Deutsche med. Wochenschr.*, No. 21, 1889.
- (185) "Beiträge zur. hypnot. Therapie." *Deutsche Medizinal-Zeitung*, 1889.
- (186) *Deutsche med. Wochenschrift*, No. 46, 1890.
- (187) *Ibid.*, No. 7, 1887.
- (188) *Ibid.*, No. 19, 1887.
- (189) *The Med. and Surg. Reporter*, 1 May, 1875, and 10 and 17 November, 1883.
- (190) *Berl. klin. Woch.*, No. 30, 1890. Peltessohn gives the entire literature of the question.
- (191) "Des paralysies laryngées d'origine centrale." *France médicale*, No. 134 u. 135, 1885.
- (192) "Bulbar paralysis." *Brit. Med. Journal*, 15 May, 1886. Meeting of the Harveian Society.
- (193) "Contribution à l'étude des crises laryngées tabétiques." *Revue de médecine*, 1881, Bd. i., p. 541.
- (194) *Transactions of the Clinical Society of London*, vol. xi., 1878, p. 141. *Ibid.*, vol. xii., 1879, p. 184. *Archives of Laryngology*, 1881, vol. ii., p. 215, Case 21. *Transactions of the Clinical Society*, vol. xxii., 1889, p. 373.
- (195) "Lähmung der Glottiserweiterer als initiales Symptom der Tabes dorsalis." *Berliner klinische Wochenschrift*, No. 13, 1886.
- (196) *Edinburgh Medical Journal*, July, 1885.
- (197) *Thèse de Paris*, Oct., 1885.
- (198) *Berliner klin. Wochenschrift*, No. 49, 1885.
- (199) *Berliner klin. Wochenschrift*, No. 38, 1886.
- (200) "BRAIN," April, 1886.
- (201) *Lancet*, 18 Sept., 1886, and *Birmingham Medical Review*, Dec., 1886.
- (202) *Berliner klin. Wochenschrift*, No. 23, 1886.
- (203) *Ibid.*, No. 8, 1887.
- (204) *Ibid.*, No., 20, 1887. These cases have also been described at length by Wegener in his Thesis. Berlin, 1887.
- (205) *Archivi Italiani di Laryngologia*, Fas. 2, 1887.
- (206) *Deutsche med. Wochenschrift*, No. 26 u. 28, 1888.
- (207) *Journal de Médecine et de Chirurgie Pratiques*, Oct., 1888.
- (208) *Virchow's Archiv.*, vol. cxx., 1, 1890.
- (209) *New York Medical Record*, 21 Dec., 1889.
- (210) *Rivista Sperimentali di Phrenatria*, 1890, und *Archiv. für Psychiatrie* 1889, Bd. 21, S. 156.
- (211) *London Hospital Reports*, vol., 1864, et seq., see also *Lancet*, 1 April, 1886. *Sitzung der Harveian Society*.

- (212) *Brit. Med. Journal*, 3 March, 1883; *Trans. Clin. Soc. of London*, vol. xix., p. 317, 1886; *Brit. Med. Journ.*, 30 March, 1889, Meeting of the Clinical Society.
- (213) *Clinical Society's Transactions*, vol. xxii., p. 322.
- (214) *Medycyna*, No. 21, 1187.
- (215) *Prager Med. Wochenschrift*, No. 16, 1889.
- (216) *Brit. Med. Journal*, 14 December, 1889. Meeting of the Hunterian Society.
- (217) *New York Med. Journal*, 28 Sept., 1889. In Wright's two cases the palate was not affected.
- (218) *Proceedings of the Royal Society*, vol. 44, p. 272.
- (219) See previous footnote.
- (220) "Ueber centrale Kehlkopflähmung." *Deutsche med. Wochenschrift*, No. 21, 1886. "Zur Pathologie der centralen Kehlkopflähmungen." *Archiv. für Psychiatrie*, vol. xix., 1888.
- (221) *Deutsche med. Wochenschrift*, No. 27, 1885.
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- (226) *Brit. Med. Journal*, 2 July, 1887.
- (227) Mittheilungen aus der med. Klinik zu Würzburg, Bergmann, Wiesbaden, 1885.
- (228) "Paralysis of the lateral adductor muscle, etc." *New York Med. Journal*, 12 February, 1887.
- (229) "Two cases of unilateral adductor paralysis, etc." *Lancet*, 13 Oct., 1888.
- (230) "Étude sur la paralysie isolée du muscle ary-aryténoïdien." Bordeaux, 1888.
- (231) *Annales des Maladies de l'Oreille, etc.*, Juli, 1885.
- (232) *Ibidem*, Mai, 1890.

ON HYSTERIA,

BY J. MICHELL CLARKE, M.A., M.B., M.R.C.P.

- (1) Leçons Cliniques sur l'Hystérie et l'Hypnotisme faites à l'Hôpital de St. André de Bordeaux par A. Pitres, Professeur et Doyen de la Faculté de Médecine de Bordeaux, &c. D'une Lettre-préface de M. le Professeur J. M. Charcot. Vol. i. and part of Vol. ii. Paris: Octave Doin, 1891.
- (2) Traité Clinique et thérapeutique de l'Hystérie d'après l'enseignement de la Saltpêtrière par le Dr. Gilles de la Tourette, ancien chef de clinique des maladies du système nerveux à la Saltpêtrière. Préface de M. le Prof. J. M. Charcot. Vol. i., Hystérie normale ou interparoxystique. Paris: E. Plon Nourrit et Cie, 1891.
- (3) Les Agents provocateurs de l'Hystérie par Georges Guinon. Paris: Bureau de Progrès Médical, 1889.
- (4) L'Hystérie Male dans le service de M. le Prof. Pitres à l'Hôpital Saint André de Bordeaux par le Dr. Emile Bitot. Paris: 1890.
- (5) Les Anesthésies Hystériques des Merqueuses et des Organes de sens et les zones hystérogènes des muqueuses par L. Lichtwitz. Paris: 1887.
- (6) Des Anesthésies Hystériques par le Prof. A. Pitres. Bordeaux: 1887.
- (7) Des Zones Hystérogènes et Hypnogènes par le Prof. A. Pitres. Bordeaux: 1885.
- (8) Étude Clinique sur les troubles de la Sensibilité Cutanée chez les Alcooliques par le Dr. Edmond Grasset. Bordeaux: 1887.
- (9) Quelques cas d'Hystérie Male et de Neurasthénie par le Prof. Grasset. Montpellier et Paris: 1892.
- (10) Recherches Cliniques sur les troubles de la Sensibilité Cutanée dans la Chlorose par le Dr. P. G. Laporte. Bordeaux: 1888.
- (11) Recherches Cliniques et thérapeutiques sur l'Épilepsie, l'Hystérie et l'Idiotie par Bourneville, médecin de Bicêtre. Paris: 1890.
- (12) Comparaison des effets de divers traitements dans l'Hystérie précédée d'une esquisse historique sur la Metallothérapie par Oscar Jennings. Paris: 1878.

THE authors of the first three books have all been pupils of Prof. Charcot, and indirectly or directly, the works are the outcome of that systematic study of hysteria, initiated and carried on by him at the Saltpêtrière, which has been productive of such great results in extending our knowledge of the neurosis, and in

putting it on an accurate scientific basis. Both Prof. Pitres' and M. Gilles de la Tourette's books are preceded by a preface written by Prof. Charcot. The direct object of the latter work is indeed to bring together, in an easily accessible form, the substance of M. Charcot's teaching on hysteria. This first volume treats only of "normal" hysteria, that is to say, the ordinary state of the hysterical in the interparoxysmal periods; in it, only the common basis or foundation of the neurosis, as evidenced in the presence of permanent "stigmata" is considered, and not the convulsive seizures or other paroxysmal manifestations, which are to be dealt with in a second volume.

The work will thus form a systematic treatise on hysteria. It is written with perspicacity and clearness, and with a completeness that shows the author's extensive knowledge of his subject; nor must we forget to mention that his own original contributions to the study of hysteria have been numerous and important. The reader will find full information on every branch of the subject considered in this volume; and the full consideration of, and abundant references to, the work and views of other observers, both French and foreign, enhance its value as a book of reference. Perhaps we may specially mention the sections on disorders of vision, hysterical tremor, the mental state in hysteria, and the interesting historical notices which occur throughout the work.

Prof. Pitres has arranged his book on a somewhat different plan. It is based on the study of one hundred cases of hysteria taken from his clinical wards. In it will be found an admirably written account of the chief manifestations of hysteria, and one of the leading features of the book is the graphic way in which cases are brought forward—presented before the reader's eye, so to speak—in order to illustrate the subject under discussion. Such a mode of treatment is a very valuable aid to the memory in helping one to get a distinct mental picture of symptoms or groups of symptoms. It is impossible, in the following digest, to do justice to this character of the book, and to the life-like manner in which the clinical descriptions are given—a branch of the medical art in which, as is well-known, the French particularly excel. There are some interesting plates at the end of vol. i., representing the cures of various (hysterical) disorders that took place in the eighteenth century, at the tomb of the Deacon Pâris. Vol. ii., which is almost entirely occupied by the subject of hypnotism, was reviewed by Dr. Lloyd Tuckey in the last number of this journal.

The scope of M. Guinon's work is more limited than that of the other two, it has already been reviewed in *BRAIN*, vol. xii., p. 407, by Prof. Marie.

The following historical sketch is taken from M. Gilles de la Tourette's work. Plato first formulated the theory, which so long regulated the treatment and clinical study of hysteria; he thought that the uterus was an animal which ardently desired to engender children, and when after puberty it remained a long time sterile, it ran over the body, obstructed the entrance of air, stopped respiration, threw the body into extreme dangers, and caused divers diseases.

Hippocrates who was thoroughly familiar with epilepsy, does not appear in his work to always differentiate it very clearly from hysteria, with which he was certainly acquainted but much more imperfectly.

A passage from Celsus (1) is quoted in which this author clearly brings out the elements of the differential diagnosis between epilepsy and hysteria.

Hysteria flourished in the ignorance and religious fanaticism of the middle ages. Our knowledge of hysteria at this epoch is due to the labours of Charcot et Richet, who have in their works, "*Les démoniaques dans l'art*" (1887), "*Les malades et les difformes dans l'art*" (1889) and "*Études Cliniques sur la grande hystérie ou hystéro-épilepsie*," brought together a number of documents and pictorial representations of hysterical persons. One of the oldest is a miniature on the manuscript of the Emperor Otto preserved in the Cathedral at Aix-la-Chapelle, which represents the young man possessed with devils being brought before Christ; the patient is represented in the midst of an hysterical convulsion with the trunk forcibly bent backwards. A bas-relief in bronze from the gate of a church at Verona, represents a woman possessed with a devil; under the benediction of the bishop to whom she has been brought, the devil is represented, as going out of her mouth: the woman herself is bent backwards in form of the "*arc de cercle*" with a prominent and tympanitic abdomen. Both these examples date from the 11th century. There is a still earlier (5th century) representation of an exorcism in which the person possessed is in an hysterical attitude. Dr. de la Tourette notes that the possessed, *i.e.*, the hysterical persons were in two of these instances males.

The extensive epidemics of chorea of the middle ages were not true chorea, but rhythmical hysterical chorea—witness the representations by Breughel in 1574 of the "*Dancers of St. Guy*,"

which M. G. de la Tourette reproduces from "Les Démoniaques dans l'Art."

In 1618 Charles Lepois of Pont-à-Mousson anticipated many of the modern views upon hysteria. He recognized the existence of hysteria in children and in men; he gave a description of the sensory troubles premonitory of an attack, *i.e.*, obnubilation of sight and of hearing, loss of voice, and of the salivation following the attack; he recognized the paralysis of the upper and lower extremities. Sydenham's observations on the neurosis were the best and most complete up to the present century. The accuracy of his work was, however, not understood by his contemporaries, nor appreciated at its true value until the labours of Briquet in 1859. "With the eye of genius Sydenham had seen so many things which appeared so singular and incomprehensible at their first description, that he remained entirely misconceived." With the exception of Sydenham the authors of the 18th and of the first half of the 19th century lost themselves in the maze of empty and fruitless discussions which had a disastrous result on the evolution of the scientific knowledge of hysteria.

The work of Louyer Villermay (2) in 1816, denied the existence of male hysteria, and stated that the most frequent causes of hysteria were the deprivation of the pleasures of love, the chagrins arising from this passion, and menstrual irregularities. His views, as Briquet said, belonged to 1500 rather than to the year 1816. They were ably combated by Georget. The French writers about 1846, Brachet (3), Landouzy (4), do not seem to have been acquainted with the masterly lectures of Brodie (5) written 10 years previously. Brodie admits male hysteria, and in describing hysterical coxalgia says: "It is not the muscles which do not obey the will but the will itself which does not enter into action." He was the first to give the proper method of treatment for hysterical paralysis and contractions.

In 1859 appeared "Briquet's Treatise on Hysteria," which contains an immense amount of accurate clinical observation on hysteria. He concludes that the disease ought to be considered as a functional affection (*affection dynamique*). The various phenomena of hysteria are very exactly treated, but the general laws on which the complex symptoms depend are not mentioned, and their discovery, which has so revolutionised the knowledge of hysteria, dates from the labours of Professor Charcot at the Salpêtrière. Previously the differences between hysteria and epilepsy were ill defined, and it was thought that hysteria and epilepsy could be combined in the same patient as hystero-

epilepsy. Charcot showed that they are never combined, though they may exist separately in the same patient. Hystero-epilepsy contrary to appearances, is nothing but pure hysteria in its most developed form—*hysteria-major*. He retained the term *hystero-epilepsy*, applying to it this new meaning. In 1873 he described the hysterical convulsion as following certain laws which were enunciated for the first time. He also pointed out the existence of the “*status hystericus*” (*état de mal hystérique*) as opposed to the “*status epilepticus*” previously known. He described hysteria as a disease “one and indivisible” and gave clear indications for its diagnosis from epilepsy. To him we owe also the study of the permanent “*stigmata*” of the neurosis, the presentation of hysteria in the male from an entirely new point of view, and the working-out of the chemical phenomena of hysteria and of traumatic hysteria.

M. Pitres observes that in the middle ages the absorbing character of religious ideas, the frequent pictorial representations of demons, the burnings of persecution determined the form of hysteria; the patients were “possessed of devils.” In our days the environment is changed, the dominant ideas are of another kind, and belong to the side of the affections, the affections, *e.g.*, contrarieties in love are more apt to produce hysteria. There has been no change in type of the neurosis; the “possessed of devils” and the patients of to-day both show the same disorders of sensation, contractions, paralyses and convulsions. Even now epidemics of “demoniacal” hysteria occasionally develop in remote districts and are very instructive. The epidemic of Morzine, a remote mountain village in the Haute-Savoie, where the people are illiterate and very superstitious, is a good instance. In 1857 a young girl saw her companion taken out of a stream half drowned, she fell down without consciousness, “as if dead.” A few days later a friend who was with her in one of these attacks was similarly affected. The two girls afterwards had convulsive seizures, followed by periods of hallucination, in which they blasphemed horribly, uttered prophecies—some of which were verified—and climbed trees like monkeys. In the course of 8 months, 18 people were affected. Three patients were cured by their fathers threatening them with severe punishment if they continued to be convulsed. This being the state of affairs the ecclesiastical authorities held public exorcisms. The malady now rapidly spread and in 1861 about 120 people had had convulsive attacks. Some persons who were sceptical as to the alleged demoniacal origin of the fits were denounced as heretics, and one

barely escaped with his life from the popular fury. The epidemic was finally stopped by the government sending a force of *gend'armes*, removing the parish priest, isolating the patients, and removing the worst cases to distant hospitals. It is unnecessary to point out the many analogies between this modern outbreak and those that are on record as occurring in the middle ages.

M. Pitres begins his work by enumerating five propositions which sum up the characters common to the manifestations of hysteria and serve as a basis for a clinical definition of the disease as a substitute for the nosological definition which is at present impossible.

(1) Hysterical phenomena result from purely functional disturbances of the nervous system.

(2) They may be abruptly excited, modified, or suppressed by psychical influences or physical agencies which have no effect on similar phenomena due to organic disease.

(3) They very seldom exist alone; in the immense majority of cases certain latent stigmata coexist with the conspicuous manifestations of the neurosis.

(4) They have no regular evolution; they come on in no pre-established order and proceed under different forms and at different times in the same subject.

(5) As a rule they have not that profound effect on the general health and mental state of the persons affected by them that similar phenomena dependant on another cause would have. The author lays especial stress as of real diagnostic value on the indifference and serenity of hysterical women in the presence of the various illnesses from which they may be suffering. They never believe themselves seriously ill and they never lose their self-consciousness, their regard for their appearance, &c.

The records of former centuries especially from the XIIIth. to XVIIth., when hysteria often took the form of epidemics prove that it is not a disease caused by our high state of civilisation.

The primary cause of hysteria, M. Charcot teaches, is heredity; this may be similar as when mother and daughter both suffer from the neurosis, or it may act by transformation, as in cases where the progenitors of the patient have suffered from some nervous disease other than hysteria. Apart from heredity, there exist only the "exciting" causes of the disease.

Briquet (6) found in 351 hysterical patients, whose families comprise a total of 1,103 people, 430 men and 673 women, that amongst their antecedent and collateral relatives 214 were hysterical, 13 epileptic, 16 insane, 1 had delirium tremens, 1 was

paraplegic, 3 somnambulists, 14 affected with convulsions, and 10 with apoplexy. Twenty-five per cent., therefore, of these 1,103 people suffered from affections of the nervous system. Persons born of hysterical parents are twelve times more predisposed to hysteria than those whose parents are non-hysterical. Sometimes hysteria, from the number of patients affected in one family, takes almost the character of a "family" disease. Hammond (7) states, that in 209 cases of hysteria where he had enquired into the question of heredity, 131 patients had mothers, grandmothers, or aunts affected with this malady, and in many others, the parents suffered from other nervous diseases. It is difficult to reconcile this statement with that of Russell Reynolds: (8) "Hereditary taint has not been shown to exert any marked influence in the development of hysteria." As regards male hysteria, Batault, (9) in his inaugural thesis compiled in the clinic of M. Charcot states, that of 100 cases of hysteria in men, a pathological heredity was traced in 77, and did not appear in the 23 others. The above 77 cases came from 75 families, there being two instances where two brothers were affected. In 56, one or other of the parents, or both, were affected with some nervous taint. The mother alone was affected in 35 cases, both parents in 12, and in 9 the father only. Thus, in both male and female patients, it is the mother who is the most often nervous.

Bodenstein, (10) in 11,225 cases of nervous disease in the clinic of Profs. Mendel and Eulenberg, found 1,224 hysterics, of whom 122 were men. He says "Heredity is shown still more often in men than in women." Bitot, (11) from the clinic of Prof. Pitres at Bordeaux, in 22 cases of male hysteria, traced a neuropathic heredity in 17, in 2 others the fathers were intemperate.

The influence of heredity, here direct heredity, is above all, marked in children; in 58 of 80 cases, varying from 1 to 12 years of age, the parents were hysterical, 2 were insane and 3 epileptic. (Briquet.)

Are such morbid agencies as syphilis and alcohol to be considered as exciting or primary agents; and are "arthritis" and tuberculosis in the parents capable of engendering hysteria in the offspring? M. de la Tourette thinks that arthritis, alcoholism, and syphilis in the parents only act by bringing out in the children the hereditary nervous taint which remains latent in the parents; and that outside the neuropathic group of diseases hysteria has no connections. At the same time, M. Charcot has shown that the uric acid diathesis, arthritis, syphilis, and tuberculosis, are often associated with the neuro-

pathic diathesis. "A neuropathic hereditary tendency is thus the primary cause of hysteria; the neurosis is a branch of the great neuropathic family. Its connections with arthritism, for example, are only those of contact and not of fusion. Hysteria is one in its heredity as it remains indivisible in its evolution."

In 272 cases of hysteria in children the onset occurred:

				GIRLS.	BOYS.	TOTAL.
Below 3 years in				19	1	20
At the age of 3 years				—	1	1
"	"	4	"	1	1	2
"	"	5	"	4	2	6
"	"	6	"	3	2	5
"	"	7	"	15	4	19
"	"	8	"	16	6	22
"	"	9	"	15	7	22
"	"	10	"	18	15	33
"	"	11	"	24	17	41
"	"	12	"	22	13	35
"	"	13	"	27	16	43
"	"	14	"	12	8	20
"	"	15	"	—	3	3
				176	96	272

The proportion of girls to boys is a little less than 2 to 1.

The influence of the appearance of menstruation in girls thus appears to be nil.

Women.—From statistics based on his own observations and on those of preceding authors, Briquet concluded (i.) one-fifth of cases of hysteria in women develop (in France) before the age of puberty; (ii.) rather more than a third of the cases develop from 15—20 years; (iii.) the frequency of hysteria rapidly decreases from 20—25 years of age, being only half as frequent as in the antecedent period. From 25—40 it remains at the same proportion, being ten times less frequent than from 12—20 years of age; (iv.) hysteria is very rare between 40 and 60 years and is not more frequent at any one year than at another during this period.

Men.—M. Batault shows that the date of the first appearance of symptoms in men is most often between the ages of 10 and 20 years. Of 192 cases of male hysteria in 44 cases the onset was between 10 and 15, and in 34 cases between 15 and 20. The predisposition due to age conforms much less to the period of

development of the genital organs than is generally supposed. M. Gilles de la Tourette thinks, judging from his experience at the Saltpêtrière, that the time of onset is later in men than in women, which is accounted for by the greater incidence of exciting causes, such as traumatisms and intoxications, during adult age in men. In 22 cases of hysteria in men reported by M. Bitot (12) the mean age of onset was 26 years.

What is the real frequency of hysteria? Sydenham wrote that as "fevers with their attendants constitute two-thirds of the diseases to which mankind are liable, upon comparing them with the whole tribe of chronic distempers, so hysteric disorders or at least such as are so called make up half the remaining third part, that is they constitute one moiety of chronic distempers."

With regard to the frequency of hysteria in man and its proportion to the number of cases in women, M. Bodenstein in 11,225 cases of nervous disease observed 1,224 hysterics of whom 122 were men or 1 to 10 women. This is double the scale of Briquet, who said that the proportion was 1 to 20.

In 100 cases of hysteria Pitres found 31 men and 69 women, or about 1 to 3.

M. P. Marie (13) while at the "Bureau central d'Admission," where patients make application for admission to hospital, examined during May, 1889, 697 persons affected with various diseases of whom 172 were women and 525 men. He sought to determine the frequency of hysteria in the two sexes, and to that end divided the patients who presented symptoms of hysteria into two classes (i.) "hystérie massive," those in whom there was well marked anæsthesia, general, unilateral, or limited to a limb, and ii. "hystérie mitigée," in which symptoms of hysteria were certainly present but not accompanied by disturbances of cutaneous sensation. Of the 525 men, 251 presented the characters of "hystérie massive," 3 of "hystérie mitigée," of the 179 women one only fell in the first, five in the second category. The percentage of cases in the two sexes of "hystérie massive" is therefore 4.76 per cent. men, 0.58 per cent. women. The male patients belong to the working classes, or those who have descended in the social scale and have therefore very frequently been exposed to the exciting causes of male hysteria, traumatisms, poisoning by alcohol, lead, &c. The social status of the women is a little higher and they are therefore less exposed to the action of the above causes. M. Maires thinks that the explanation of this very unexpected proportion between the two sexes lies in the fact that male hysteria is much more rare in the middle and upper classes

as compared with the lower, whereas this is not the case in hysteria in women. As regards the second class "*hystérie mitigée*," there is 1 case in 35 women and 1 in 175 men: this form is therefore much more frequent in women, and this is a fresh confirmation of M. Charcot's teaching that hysteria in men is generally characterised by its obstinacy and severity.

M. Souques (14) compiled the statistics of 441 men and 240 women under the care of M. Chauffard at the Broussais hospital and found amongst them 26 cases of hysteria amongst the men and 6 cases only amongst the women.

M. Gilles de la Tourette gives similar statistics derived from the records of the Salpêtrière, where the patients come from all classes and from all countries. During the two years from November, 1887 to November, 1889, there were, in the wards under his care as "*chef de clinique*," 323 patients, comprising 200 women and 123 men, of whom 67 women and 37 men were suffering from hysteria, in the proportion of 2 to 1. In the out-patient department of 726 patients of both sexes, there were 51 female and 21 male cases of hysteria or again about 2 to 1. M. Parinaud, the ophthalmic surgeon to the hospital, examined 49 women and 30 men affected by visual disturbances due to hysteria, but this great proportion of men is partly accounted for by the fact that many of the hysterical women, in whom the symptoms were well marked, were not sent to him for ophthalmoscopic examination, whilst all or nearly all the men were examined, and that a large number of cases of male hysteria were especially sent to M. Charcot from the different hospitals, on account of the interest he took in them. But even allowing for this, the number shows the frequency of male hysteria. (15) At the Salpêtrière hospital, therefore, where all social grades are represented, one case of hysteria in man occurs for every two or three in women.

M. Pitres attaches considerable importance, from the point of view of prognosis and prophylaxis, to the neuropathic symptoms shown in early childhood by those who are liable to become hysterical. Such are cataleptic attacks, suffocative attacks, certain varieties of cough, which resemble whooping-cough and last for weeks or months; attacks of hiccough or vomiting without sufficient cause, palpitation, night-terrors, headache during adolescence; certain forms of pulmonary congestion, of hæmorrhage from the nose, stomach, or lungs; somnambulism, contractions of the limbs, or temporary paralysis of motion or sensation, neuralgias, and pains in the joints.

Exciting causes of Hysteria :—

(i.) Strong moral emotions. Instances of emotions as exciting causes of hysteria abound. Fear is the most potent. Sometimes a single moral shock suffices to evoke hysteria, often it only appears after a longer or shorter series of repeated emotions. From Brodie to the present time all authors are in accord as the evil influence in this direction of an injudicious bringing up. M. Guinon instances first corporal punishment, or physical ill-treatment. Blows may act with double force provoking symptoms of hysteria by moral shock, and by traumatism. Religious practices carried to extremes are often the starting point of hysteria.

Equally effectual are the pernicious practices of deterring children from doing wrong by threatening them with a bogie or a policeman, and of allowing them to be constantly reading stories in which sorcerers and ghosts play prominent parts. M. Charcot (16) relates the very instructive history of an epidemic of hysteria which took place in a family much given to spiritualistic séances. The obvious lesson is to carefully protect children who are nervous and impressionable from all such influences.

(ii.) Alongside of the moral emotions we must place imitation as an exciting cause, the influence of which is well known in epidemics of hysteria. M. Charcot considers that the form of hysteria which is developed in a patient by imitation of the symptoms seen in other hysterics is not a very grave one, and is fraught with less evil to the future welfare of the patient than other forms of hysteria.

Attempts at hypnosis constitute one of the most effectual agents in the causation of hysteria. The convulsive attack is commonly the symptom which follows on injudicious hypnotisation. M. Guinon narrates a case in which the only hysterical manifestations were slight cough and hiccough, and in which extremely violent and frequent hystero-epileptic convulsions followed repeated unsuccessful attempts at hypnosis. He thinks that hypnosis should never be resorted to except in cases where the neurosis is present in so severe a form that there is no risk of making the patient worse.

Influence of traumatisms on the development of hysterical symptoms.—M. Guinon observes on this subject, that the name of “nervous shock” is applied to that state in which a person falls who has been the victim of a traumatism or of a more or less violent physical shaking, accompanied as it always is by strong emotion. It is a state in which the symptoms are partly physical,

partly psychical. By traumatic hysteria, we mean hysteria developed under the influence of an injury, and the accompanying nervous shock. Nervous differs from what is called traumatic "shock" (shock following severe operations and injuries). The latter is always a grave accident and often fatal, while the "former does not present any gravity in itself, at least in so far as regards the life of the patient."

The knowledge that such symptoms follows an injury dates from Erichsen's work. (17) The cause of the symptoms was considered by him to be an inflammation of the spinal cord and its membranes.

In 1882 appeared the first edition, the second followed in 1885, of Page's work on "Injuries of the spine and spinal cord and Nervous Shock:" in this book the brain was considered to be the primary seat of the functional troubles observed, and the term "Railway-Brain" was substituted for "Railway-Spine," the principal factors of the condition being neurasthenia and *hysteria*. The next step forwards came from America when Mr. Walton, (18) returning from the study of nervous diseases at the Saltpêtrière, attributed the various nervous symptoms that follow on railway and other accidents to hysteria.

In Germany the question took another aspect. Oppenheim and Thomsen (19) from the study of the symptoms following accidents, wished to establish a special neurosis, the traumatic neurosis, from the presence of such symptoms as hemianæsthesia, sensory anæsthesias, contraction of the visual fields, &c., from the obstinacy of the hemianæsthesia, as opposed to its variability in hysteria, and from the mental state of the patients. M. Charcot (20) however showed that hysteria is often provoked by a traumatism, and that the majority of the nervous symptoms recognised in the injured are in reality only hysterical symptoms identical with those present in hysteria from whatever cause it may arise.

The hypothesis of a distinct traumatic neurosis was further brought forward in a modified form by MM. Oppenheim, Strümpell and others, but the question seems now to be finally settled that the symptoms are due to hysteria, "traumatic hysteria;" this result is largely due to the labours of Charcot and his pupils. The author thinks that the cause of the confusion is, in great part, to be attributed to the prevailing ignorance of the symptoms of male hysteria.

M. Pitres observes that the effects of traumatisms differ according to the morbid predispositions of the subjects; in one

exciting an epileptic discharge, in another neurasthenia, in a third hysteria, and that there is no reason to separate hysteria arising from this cause from hysteria due to other causes, *e.g.*, moral emotions. The formation of a "traumatic neurosis" would be a false step in neurology, as it would be an artificial classification depending upon etiological factors only.

The facts are too numerous and too well authenticated to allow it to remain doubtful that the connection between traumatism and hysteria is one of cause and effect (in a predisposed subject) and not of mere coincidence. In further support of this may be adduced the fact that hysterical manifestations provoked by one traumatism, may be caused to disappear under the influence of another one. Cases of traumatic hysteria are of great importance from a medico-legal point of view. Railway accidents account for a good many of them, but other causes are equally efficient, *e.g.*, the fall of a mason from his scaffold, (21) of a coachman from his box, (21) a bronze-worker gives himself a blow on the fingers with his hammer, (21) a mother strikes her son on the ear. A burn, the application of a fracture apparatus, the wound inflicted in an operation, or even the force exerted in holding the patient in a particular position during the operation, have all been found effectual causes. Earthquakes, from the over-mastering terror they excite, are potent causes of hysteria; in the same way, lightening-stroke, or the fright produced by the near fall of a thunderbolt, may give rise to the neurosis. From numerous examples, M. Guinon concludes that the nature and degree of severity of the injury have little influence on the development of hysterical symptoms, and that any kind of injury whatever may provoke hysteria. For instance, in railway accidents—and the same remark applies to earthquakes—those who are most severely injured are not necessarily the most liable to consecutive hysteria. The contrary is almost true. The patient who afterwards becomes hysterical, is often able to reach his destination, either by another train, or sometimes even on foot. Meanwhile, the traumatism has indisputably a direct action on the determination of certain symptoms of local hysteria, *e.g.*, a fall on the shoulder has caused an hysterical paralysis of that arm. M. Pitres says, with regard to this point, that although there is a casual connection between a traumatism and the contracture which follows it, this contracture is not to be considered as a mere local effect of the traumatism, a so-called "local or peripheral hysteria." The injury has not caused the hysteria since that is present before and after the accident, it has

simply determined in the evolution of a malady already in existence a symptom which without the injury would never have appeared.

Prof. Pitres sums up the clinical characters of hysterotraumatic monoplegias in the following terms :—

(1) They occur indifferently in men and women, rather more frequently in the former.

(2) The patients are generally between the ages of 20 and 50 (adult life).

(3) They generally appear in persons predisposed to hysteria by hereditary influences, and who have previously suffered from neurotic affections. This, however, is not always the case, and a traumatic monoplegia may be the first manifestation of hysteria in a person previously free from it.

(4) The paralysis bears no relation to the severity of the injury. It follows severe or slight injuries, of local or general incidence, whether they are accompanied or not by nervous shock or emotional disturbance. A trifling accident may be followed by a paralysis which persists for years, whilst one that follows a serious injury may last for some days only.

(5) The paralysis may sometimes develop immediately after the accident, but usually comes on after an interval of hours or days.

(6) The paralysed muscles are flaccid ; the electrical reactions are normal ; the tendon-reflexes are normal, sometimes a little exaggerated. The muscular sense is lost. Rapid wasting often supervenes in the muscles affected.

(7) The skin of the paralysed limb is generally anæsthetic or analgesic. This anæsthesia may be limited to the limb, or extend to the corresponding half of the body, or may be general.

(8) The patients often, but not always, present the permanent stigmata of hysteria, *e.g.*, concentric contraction of visual fields— anæsthesia of pharynx. There are sometimes convulsive attacks and hysterogenic zones. They cannot usually be hypnotized.

(9) The duration of the paralysis is very variable. Cure is certain, but at an indefinite time. It often supervenes on a moral cause.

Toxic Hysteria.—In poisoning by lead, mercury, alcohol, bisulphide of carbon, &c., hysterical manifestations are observed, more commonly in the chronic than in the acute forms. The recognition of the occurrence of hysteria under these conditions is of recent date. Although observations which proved that hysterical symptoms occur in plumbism, had been brought forward by

Petit (22) and by Maricourt, (23) who published a case of saturnine hemianæsthesia in which the absence of any organic lesion was confirmed by the autopsy, all authors—probably misled by the then prevailing idea that hysteria did not occur in the male—continued to attribute the anæsthesias of the general and special senses found in plumbism to organic changes in the nervous system produced by the poison, until a series of observations by many writers showed that these so-called saturnine anæsthesias could be made to undergo “transference” by the action of the magnet or faradic current. M. Charcot demonstrated clearly the hysterical nature of these anæsthesias, and, at the same time, the frequency of hysteria in men. (24) Poisoning by lead, alcohol, mercury, &c., may aggravate a pre-existing hysteria, or determine the advent of symptoms in a person predisposed to the neurosis, but hitherto free from symptoms. The poisons of the specific fevers, of diabetes, chlorosis, and syphilis may act in the same way. Plumbism, alcoholism, hydrargyrisim, &c., may give rise to two groups of symptoms, the one directly due to the action of the poison; the other, of hysterical nature, and resembling those of ordinary hysteria. The former are directly produced by the poison, and soon cease when the patient ceases to be exposed to its action; the latter, once excited, run an independent course, and may disappear, (whilst the action of the poison still continues) being modified or suppressed by moral emotions or physical causes, just as in other forms of hysteria, or on the other hand, may continue after the poison has been eliminated from the system.

For instance, a painter who had had two years previously, an attack of lead poisoning was admitted suffering from colic, vertigo, headache, and paresis of the right upper extremity of 14 days' duration. There was a blue line on the gums; the abdomen was retracted and tender: two doses of castor oil removed the abdominal symptoms. The affection of the right arm, which consisted in general feebleness of movement, much more marked in the extensors than in the flexors (the wrist could not be fully extended), together with anæsthesia over the inferior third of the forearm, the dorsum and palm of the hand, and over the thumb, persisted. The diagnosis was doubtful but was cleared up by the application of a magnet to the limb when the motor and sensory symptoms disappeared completely in half-an-hour.

M. Pitres directs attention to the frequent occurrence of anæsthetic patches in poisoning by lead and alcohol. The clinical characters of this cutaneous anæsthesia are identical with that of

hysteria. This anæsthesia may be either complete or partial. Its distribution may be general or unilateral but most commonly occurs in disseminated patches. In the lower extremities it generally affects the feet and the legs enveloping them in anæsthetic boots, so to speak. In the upper extremities it often spares the palms and forms larger or smaller bracelets surrounding the limbs. On the trunk it frequently occurs "en cuirasse." The patches are often asymmetrical, their borders are clearly defined and their distribution shows no relation to that of the sensory nerves or the arteries of the skin. Neither the nutrition nor the temperature of the skin are affected and there are no vaso-motor disturbances. There exist also analgesia or anæsthesia of the mucous membranes of the mouth, pharynx, larynx, nose, ears, with general or partial impairment of taste, smell, and hearing and concentric contraction of the visual fields.

These anæsthesias correspond to those of hysteria in the absence of any organic lesions in the peripheral nerves or nervous centres, and in their tendency to disappear suddenly in consequence of moral emotions, electrification, or application of metals (æsthesiogenic agents).

With regard to alcohol, Petit (25) who first makes mention of symptomatic hysteria, especially named alcohol as a cause of the neurosis. Nearly all authors consider that alcoholism is only occasionally a cause of hysteria, but M. Guinon observes that in making this statement most of them leave out of account those cases in which the neurosis formerly existed and was again excited into fresh activity by alcohol. With regard to one form of alcoholism Lancereaux states that the convulsive attacks from which absinthe-drinkers suffer are hysterical and not epileptic, and that he has never seen true epilepsy excited by drinking absinthe.

Two nervous disorders attributed to the action of mercury M. Guinon thinks are hysterical, namely, the sensory troubles and the convulsive and paralytic phenomena. The most important observations on hysteria provoked by mercurial poisoning are due to M. Letulle. (26)

M. Guinon prefers the terms hystero-saturnism, hystero-alcoholism, hystero-hydrargyrisms for these affections, because no theory is implied by them and they do not therefore lead to misconception of the true nature of the cases. M. P. Marie (27) first pointed out the occurrence of hysterical symptoms in cases of poisoning by bisulphide of carbon.

In the second of his two works on the subject, Delpech (28)

divides the course of the symptoms caused by this form of poisoning into two stages. The *first* is characterized by cephalalgia, violence of character, epileptiform convulsions, sexual excitement and hyperæsthesias. The *second* by intellectual apathy, amounting even to hebetude, sad and terrifying dreams, cutaneous anæsthesias and affections of the special senses, impotence, and paralyses. M. Guinon shows, from the descriptions of recorded cases and from his own observation, that though neuritis occurs and causes certain nervous troubles, the nervous symptoms apart from these, are hysterical, and relies for proof of this on the characters of the anæsthesias, paralyses, affections of special senses, presence of hysterogenic zones and mental symptoms.

The first attack of hysteria may occur after or be provoked by anæsthetisation by chloroform, and this has occurred in cases where dread of the operation and the emotion produced by waiting for it can be eliminated.

The case is quoted (29) of a man who felt no disturbing emotions before the operation, which was a trivial one, in fact he wished for the operation and had already previously undergone without inconvenience a similar one, but on beginning to recover from the chloroform he had a violent hysterio-epileptic attack lasting three quarters of an hour.

Besides symptoms due to organic disease of the central nervous system or peripheral nerves, poisons may evoke purely functional disturbances, in part belonging to hysteria. For Petit (30) hysteria is "symptomatic" of alcohol, lead, anæmia, &c. This theory of the "symptomatic hysterias," which is supported by Debove, (31) supposes that lead, alcohol, mercury, &c., are hysterogenic poisons, and may directly produce hysteria, complete in all its forms, even in persons not predisposed to it. They compare the "toxic hysterias" to the "toxic epilepsies," each of which has its characteristic clinical physiognomy. Fascinating as on many grounds this view is, it is not altogether satisfactory. A person in whom epileptiform attacks have been excited by lead-poisoning is not suffering from the same malady as another person who since infancy has been subject to epileptic fits periodically repeated without obvious cause. If the former is removed from the influence of the poison the attacks cease. Instead of speaking of a saturnine epilepsy we should say more correctly that he suffers from epileptiform convulsions of saturnine origin.

On the other hand—the "hysteria of toxic origin" theory—Charcot holds that poisons may aggravate existing hysteria, or excite its appearance in predisposed persons, but that they are incapable of creating it anew.

Apropos of two cases, one of lead, the other of alcoholic, poisoning, M. Charcot observes that the two patients were hysterical before being poisoned, and that some of the symptoms belong properly to hysteria while others depend on the super-added intoxications.

It is possible that hysteria may have been excited by the development of the intoxication, but this is not enough to sanction the names saturnine or alcoholic hysteria; we should rather say hysteria in a person poisoned by lead or alcohol.

M. Pitres, after the consideration of the foregoing theories, concludes that the distinction between true hysteria of toxic origin, and hysterical phenomena due to the action of poisons is difficult. An important difference is this, that true hysteria survives the exciting cause; but once aroused, whether by moral emotion, traumatism or poison, evolves itself independently of any new excitant; on the other hand, hysterical symptoms caused by intoxications remain subordinated and closely related in their development to the etiological circumstances which have aroused them. Traumatisms and poisons more frequently provoke hysteria in men than in women.

Hysteria is a pre-existing morbid susceptibility, in virtue of which various functional disturbances can be excited by etiological factors, which have in themselves only a secondary importance. Hysteria is innate, and the heredity creates the diathesis.

(The following observations on this question of intoxication, and on the traumatic neuroses, and on the mode of evolution of the symptoms are taken from M. Guinon's work).

Amongst the authors who do not admit "traumatic hysteria," some refuse to do so because, in their opinion, too great preponderance is given to the exciting cause. They do not allow that hysteria is a syndrome which becomes symptomatic of the action of such or such an exciting agent, and here they are perfectly right. But it is a long step from this to making neuroses afresh, and calling them traumatic, mercurial or lead neuroses. Although the same group of symptoms are produced in two individuals by a different cause in each, on this theory these symptoms would constitute a different disease in each of the two. This has been done for mercury and lead poisoning. In the case of the latter, A. Westphal (32) brings together almost all the nervous troubles which are observed as the result of chronic lead poisoning, under the head of "lead encephalopathy." Amongst his cases there are three well-marked examples of hysteria. Whilst quoting Charcot's opinions on the subject, he considers these cases to be ex-

amples of an "intoxication neurosis." M. Guinon thinks that this is a singular manner of regarding the facts, and it is better to multiply the causes of hysteria, than to give credit to such a vague term as "saturnine encephalopathy."

Others will not allow the name hysteria to be given to the nervous symptoms which follow traumatism or poisoning, because, although hysterical manifestations are present, yet other symptoms also exist which do not belong to hysteria. This is a clinical fact; and the key to the whole question lies in these complex cases, for by considering them closely, the objections brought against the theories of the French School may be refuted.

It has been said, but incorrectly, of the French School, that they hold that the nervous troubles that follow a traumatism are always due to hysteria, and to nothing else (33). But the French admit that traumatic agencies may provoke the outburst of many nervous affections, *e.g.*, paralysis agitans, epilepsy, senile tremor; they say, however, that because they have been excited by these agents there is no sufficient reason to put these cases in a category apart, and make of them a separate disease, a "traumatic neurosis." One might as well classify separately those cases in which white swelling of the knee arises from injury and those in which it appears to come on spontaneously. If ten cases of hysteria were taken, it would be impossible to say, from the actual state on examination—apart from the history—which were due to traumatism, which to lead, mercury, &c., the clinical entity is in each case identical.

M. Charcot has never held that hysteria is the only result of a traumatism; on the other hand, he has recognised the co-existence of hysteria and neurasthenia; and has pointed out (34) that this latter affection is equally frequent after traumatisms and railway accidents, and refers also to cases reported by Page (35). This coincidence of hysteria and neurasthenia is the key to the whole question; it is of common occurrence, but there is nothing to show that each of these affections remains autonomous. Just as in traumatisms, so in the case of other causes of hysteria, *e.g.*, intellectual overwork and lead poisoning, hysteria and neurasthenia may occur together.

One must bear in mind, that in a patient suffering from traumatic hysteria, other symptoms may be present, due to other causes. All the nervous symptoms complained of by an hysterical patient are not necessarily due to the neurosis. In a complex case symptoms, which may be due to an organic nervous affection, must be carefully weighed.

The exciting agents of hysteria are opportune causes of it, and nothing more; they cannot create hysteria anew, but only excite it in persons who are predisposed to it. This predisposition is hereditary, as Déjèrine points out (36). M. Guinon considers "that no convincing arguments have been advanced in favour of the existence of a 'symptomatic hysteria,' or of the division of the neurosis into several varieties of hysteria. The older and more rational hypothesis of a single and autonomous morbid entity—hysteria—is sufficient to explain all the cases, if it be granted that the exciting agents of the neurosis only play into relation to it the part of opportune (occasionelles) causes. This is easy to understand, if we consider that exciting causes behave, with regard to hysteria, just as they do in other maladies, in which their rôle of occasional cause is fully recognised and beyond dispute. It may be further remarked that these multiple or symptomatic hysterias, are all identical amongst themselves, and differ in no respect from the neurosis long described and considered as a fixed morbid entity." The exciting cause may impress a certain stamp on the hysterical manifestations. For instance, a woman, who had formerly suffered from compression paraplegia from caries of the vertebræ, when under the influence of the menopause, subsequently became hysterical, and developed an hysterical paraplegia closely resembling compression paraplegia. These phenomena are explained by unconscious cerebral influences acting in the form of auto-suggestion. At the same time no constant symptoms can be traced as occurring uniformly as the result of the action of one particular cause. All that can be said is, that speaking generally, "local hysteria," *i.e.*, monoplegias, monosymptomatic hysteria, occurs more often as the result of traumatisms than of other causes.

M. Charcot has pointed out the influence of falls or blows in producing monoplegia or contraction of the limb which has been the seat of the injury; for instance, a brachial monoplegia has followed a blow on the shoulder. Especially is this relation marked in cases of injury to the region of the eyeball, in which amaurosis, blepharospasm, contraction of the visual field have followed the traumatism.

The mode of development of hysteria under the influence of exciting agencies is twofold; it may either arise soon after the action of the cause or not till much later. The development may be best studied when the exciting cause has been in action for a brief and limited period. In cases of injury, for example, an uninterrupted chain of symptoms may follow the "nervous shock"

of the injury, or these symptoms may not appear until much later, and these cases are of interest as they are less known and less common than the first kind. M. Guinon gives the case of a man who, two-and-a-half years after a dangerous fall on a staircase, developed paralytic symptoms in the legs. The importance of these cases from a medico-legal point of view is obvious. Can we say that in a certain number of these patients, hysteria, provoked by the exciting cause, remains latent until paralysis, contractions, convulsions, draw attention to it? Some symptoms of hysteria, anæsthesia for example, may exist for a long time without the patient's knowledge. But generally, in cases of traumatic hysteria this does not occur; for the neurosis is then nearly always accompanied by a peculiar mental condition, which at once attracts attention. As a rule, hysteria is not latent in these cases.

Two or more causes may act together; for instance, in an alcoholic patient, hysterical manifestations—a case is given of paralysis of the forearm from a blow on the fingers in a drinker—may be excited by a slight injury, which would not have been sufficient to produce this result if the patient's powers of resistance had not been previously lowered by alcohol.

M. Guinon adduces many instances to show that several exciting agents of hysteria may unite, and produce an outbreak of the neurosis, when one acting alone, would not have been able to do so. There are cases in which hysteria existed at some former time, in which one cause to which the patient had been exposed was not sufficient to excite fresh manifestations of it, but in which another factor acting together with the first, was necessary to produce this result. He concludes that every individual presents a resistance peculiar to himself, to any particular cause of hysteria: one cause is more effective in his case than another. In each case there is always one cause which is more effective than any other.

All the exciting agents of hysteria, however varied and unlike each other they may seem to be at first sight, may be divided into two classes, according to the mechanism by which they arouse hysterical manifestations: (1) Those cases in which the symptoms of "local" hysteria predominate; (2) those in which the hysteria is general, without any striking local manifestations. In each group, all the cases which compose it are susceptible of the same interpretation. In "local" hysteria, the character of the paralytic and sensory affections indicates their cerebral origin; in it the immense majority of the symptoms are produced by auto-

suggestion. This auto-suggestion is derived most frequently from a traumatism, and after that from emotions. M. Charcot has shown the absolute identity there is between the phenomena of local hysteria, and those developed by suggestion in the hypnotic state. Now, paralysis during hypnosis may be brought about by verbal suggestion, for instance, by telling the patient he cannot move the limb, or by traumatic suggestion, by striking with a certain force the part that it is desired to paralyse.

Clinically, an auto-suggestion may occur in the same way. M. Charcot calls attention to what he terms "local shock." When a blow falls on a part of the body, it causes a sensation of numbness, of insensibility, or even a negative sensation of absence or loss of the part struck. In the healthy, this feeling soon passes off, but in a person predisposed to hysteria, it may intensify and undergo further development; and, as a consequence of this idea of absence of the limb, all idea of movement is totally abolished. In other cases of traumatic hysteria, the exciting agent, acting by a true traumatic suggestion, inhibits at once and persistently, motor representations. The mental phenomena that result from the action of the local shock dominate everything; the ideas of the existence of movement, and of feeling in the affected limb are instantly annihilated, and, if the patient's brain is suitably prepared for it, a psychical paralysis is constituted for a longer or shorter time. These cases are rare: as a rule, some time elapses between the reception of the injury, and the first appearance of the nervous troubles. The reason, why, in some instances, inhibition is instant, while in others, a certain lapse of time is required for the idea of paralysis to implant itself in the brain, M. Guinon thinks, lies in the difference of the cerebral state in different individuals, and not on the nature of the accident. He points out a striking parallel between these clinical conditions and the effects of suggestions made in different degrees of the hypnotic state.

When hysterical accidents follow immediately the action of the exciting cause, this is almost always a traumatism or an acute moral shock, such as terror. Terror may, indeed, be considered as a sort of sensation of powerlessness, either generalised, or localised in the lower extremities. In most cases of traumatism, terror and emotion must also be taken into account.

In the other group, the occurrence of an interval or period of incubation between the shock and the subsequent hysterical paralysis, explains the special form or the particular localisation that the exciting cause may give to the hysterical manifestations.

The local shock is the point of departure of the cerebral auto-suggestion ; the cerebral process, often an unconscious one, is, first, the loss of the feeling of the part, then the inability to move it. The arm feels heavy, flaccid paralysis, or stiff, paralysis with contracture. A phenomenon of some importance is the amnesia that often attends these accidents. In a large number of cases, with or without loss of consciousness, there is amnesia, not only for all the details of the accident, but for a certain period before it. The patients, having no notion of the details, and persuaded always of the great severity of the accident, imagine terrible consequences from it ; take their imaginings for reality, and, in perfect good faith, fabricate extraordinary accounts of the occurrence ; this amnesia has, therefore, often unjustly led them to be regarded as simulators. By a similar process of auto-suggestion, M. Guinon explains the action of the poisons of syphilis, lead, mercury, &c., in exciting hysteria. Auto-suggestion plays its part unconsciously to the patient. It may even be produced during sleep. Among the victims of accidents, terrifying dreams in which the accident is reproduced, are common. M. Charcot (37) and M. Féré (38) give instances of these dreams and their potency in auto-suggestion.

Prof. Grasset (39) recognizes the unity of hysteria, under a multiplicity of forms. He considers traumatic hysteria to be a distinct clinical type or species, with its own mode of evolution and special characters, many traits of which separate it from common hysteria. In criticizing the theory of auto-suggestion, he says that if we suppose that there is originally present a functional disturbance of the brain, capable by itself of bringing about a paralysis, to invoke the agency of auto-suggestion is to introduce unnecessary complication. In many cases of traumatic hysteria, the accident bears no relation to the succeeding paralytic or spasmodic phenomena. Will anyone seriously allege that in the cases where hemianæsthesia, affection of the special senses, or other "stigmata," follow a traumatism, they can have been produced by auto-suggestion? "Those manifestations of hysteria, which only develop a certain time after the traumatism, appear to be very difficult of explanation by suggestion. . . . To explain a post-traumatic paralysis separated from the traumatism by a long interval, it must be supposed that the injury provokes the auto-suggestion of the paralysis with this proviso, that the realization of the paralysis shall only take place after a certain time." He therefore limits the action of suggestion to cases where the hysterical symptoms arise from imitation (neuro-mimesis).

In other patients again the hysterical manifestations cannot be accounted for on this theory of auto-suggestion: amongst these, local hysteria is much less common, and the production of hysteria is due to a general alteration of nutrition which entails a failure of the whole organism, and especially of the nervous system, to perform their functions aright. Every exciting cause may bring about hysteria in this way, by interfering with nutrition. Especially is this the case in hysteria consecutive to acute or chronic general maladies, in diseases of the nervous system or genital organs, in physical or intellectual overwork, in the intoxications, in depressing emotions, and in prolonged grief.

Hysteria may appear during the exhaustion that follows upon an attack of one of the acute fevers; the nervous troubles consecutive to diseases of this class present themselves for the most part in the form of paralysis. M. Guinon says, with regard to acute rheumatism, that a sub-acute attack may appear to be more serious than it really is, because the pain and swelling of the joints determines hyperæsthesia of the skin and contractions of the muscles around them and there may thus arise a subsequent "pseudo-rheumatism" which may persist for months or even years and then disappear spontaneously (40). Diabetes may not only cause a recrudescence of hysteria but may evoke the symptoms for the first time. Syphilis may act in the same way. The various forms of anæsthesia which occur in cases of syphilis seem to be often due to consecutive hysteria (41). Malaria may impress a particular character upon hysterical manifestations, under its influence they take an intermittent form, recurring at regular intervals (42).

The old theories that placed the seat of hysteria in the genital organs are to-day universally discredited. Disease of these organs does not predispose more than that of any other organs to hysteria. Apart from this, from the importance attached to disturbance of the functions of the genital apparatus by women, a strong moral effect may be produced.

Severe hæmorrhages may be exciting causes, so may masturbation and venereal excesses from the nervous exhaustion they give rise to.

Hysteria may follow on physical or mental overwork, the latter is especially potent in adults, in children its influence is, to say the least, doubtful. Children cannot be forced to perform an injurious amount of intellectual labour. If children are given an excessive quantity of work, they do what exceeds their intellectual powers like machines, materially, so to speak; not seeing the

immediate interest or object of the task allotted to them, they are incapable of voluntarily exciting that state of over-action or excessive tension of the cerebral tissues necessary to produce an amount of work beyond their powers. If they suffer it is from too long detention in class-rooms and from want of sufficient exercise and fresh air.

Hysteria may also be excited by nervous diseases, tabes dorsalis, Friedreich's disease (43), idiopathic muscular atrophy, and pressure paraplegia from caries of the vertebræ.

In men hysteria affects especially the lower classes, whether they are unskilled labourers, or engaged in some special occupation; very often affected are stokers, engine-drivers, divers, tobacco employés, sailors, and soldiers. In the last the severe type of hysterical convulsions, absolutely analogous to that which occurs in women, has been described (44).

M. Colin observed that cases of hysteria are not at all common amongst the strictly criminal classes, and from the point of view of number, there is in them no difference between the sexes. Hysteria is much more frequent amongst tramps, beggars, and vagabonds (45).

M. P. Marie has published statistics to show that compared with men, the subjects of hysteria in women stand higher in the social scale. M. Gilles de la Tourette thinks that such statements are very difficult of exact proof. If hysteria is more common in town than in country women, it is to be attributed to the fact that the exciting causes, particularly the forms of intellectual and emotional excitement, which make so much more profound impression on the female than on the male nervous system, are more common in town life. The opinion of Briquet (46) is quoted with approval that "whatever tends to weaken the general health to a certain degree increases at the same time the excitability of the nervous system. Hence it follows that the sedentary professions, those which entail long hours in workrooms, and those which afford a precarious livelihood, in so far predispose to hysteria, whilst on the other hand the professions which necessitate movement and open air exercise, and afford at the same time sufficient means of sustenance will in equal degree be hostile to it.

Continence or excessive sexual intercourse seem *per se* to have little effect in evoking hysteria, but the hard conditions of a prostitute's life place her under favourable circumstances in other respects for the development of the neurosis.

All races are subject to hysteria. Of the white races, the Jews are especially prone to suffer from it. M. Gilles de la

Tourette says that the many observations that have now come from authors of both countries and from the large international practice of M. Charcot permit us to say that hysteria like other nervous diseases is exactly the same in Germany and in England as in France, in contradiction of the statements that have been made that hysteria is more rare in England and Germany and occurs in a less severe form than in France, and that the descriptions of M. Charcot only apply to the latter country. He quotes some observations of M. Aruch at the Veterinary School of Milan in which various nervous symptoms in three dogs, namely, paresis, paraplegia without affection of bladder or rectum, anæsthesia, aphonia, and convulsive attacks without loss of consciousness were attributed to hysteria, no lesions being found at the autopsy which could account for these symptoms.

The only way for us to get a thorough understanding of hysteria, is to study it by the experimental method. The classification of cases into acute and chronic is unsatisfactory on account of the variable and fortuitous course of the malady, regulated as it is by external agencies. The division into acute and chronic moreover ignores the latent predisposition always present. Based on the nature of the predominant symptoms in each particular case, hysteria has been classified as (1) convulsive or non-convulsive, (2) peripheral or visceral, (3) mono- or polysymptomatic. These divisions may be adopted for convenience if it is always remembered that they do not correspond to different forms of the disease. Any case by virtue of the hysterical diathesis may at one time or another present any of the various symptoms. It is exceedingly rare for one symptom to exist alone. Alongside of the symptom that has attracted the patients' attention, the physician connotes the presence of latent symptoms or stigmata, whose concomitance is indispensable to the diagnosis. Classifications based on age and sex have no value. To sum up, hysteria is one and indivisible. Under the manifestations which vary in accordance with the infinite variety of contingent circumstances, lies always the same disease as the true source of them.

In hysteria, as in all general diseases, there is a ground work on which the symptoms which constitute the affection evolve. This ground work "the general hysterical state" is formed by the union of a great many elements. It is not always complete, many links in the pathological chain may be absent at the time of observation, but one character, *tenacity*, is common to all, and they have therefore been named by M. Charcot the "permanent

stigmata" (47). Their study is of the first importance in diagnosis, for they enable one to affirm the existence of hysteria when the more striking phenomena, which are often temporary, are absent, and a thorough acquaintance with them is indispensable for a sound interpretation of these latter symptoms.

The history of the discovery of anæsthetic patches in hysterical subjects is very interesting, M. Pitres recalls that when the magistrates who were investigating a case of witchcraft examined the accused, they began by trying to establish moral proof of demoniacal possession by questioning him on his family history and his antecedents, and endeavoured to get him to confess his commerce with Satan, and then proceeded by means of pricking him with pins (the accused being blindfolded) or sharp splinters to determine the presence of anæsthetic areas in the skin, which were called *stigmata diaboli* or witch-marks. To the discovery of these stigmata great importance was attached as a proof of the transport of the accused to the witches'; "sabbath," and they were attributed to direct contact with the devil. Readers of contemporaneous accounts of trials for witchcraft will remember the importance attached to the presence of wens or moles and of small areas into which a sharp needle or awl could be thrust even to the bones without pain and at the same time without causing bloodflow. The doctors of the time shared in these superstitions, and Jacques Fontaine, "conseiller et médecine ordinaire du roy et premier professeur en son Université de Bourbon en la ville de Aix," wrote a book on "Witch Marks and the real possession by the devil of the bodies of men." (48) He thought that these "dead-anæsthetic patches" were made with a hot iron and a certain ointment, and accounted for the absence of any scar by supposing that "Satan was such an accomplished operator that he could apply the iron to the body without producing a scar." To day the physician proceeds in the same way as the judges of the middle ages; when he suspects a person of hysteria he begins by trying to obtain moral proof of the existence of the neurosis in the morbid tendencies—hereditary and personal—of the patient, and investigates the nature and character of past and present symptoms. Lastly, he searches for impairment of the general sensibility or of the special senses, which are now considered as "marks of hysterical possession" or "hysterical stigmata."

It is a remarkable fact that when witchcraft ceased to be believed in the existence of these anæsthetic patches was completely forgotten, and it is hardly fifty years since anæsthesia was recognised by doctors as common in hysteria. The first regular

observations were those of Piorry in 1843. Briquet in his work on hysteria in 1859 investigated and described them with remarkable thoroughness and perspicacity, but hysterical anæsthesia was not generally recognized until the popularization of the subject by M. Charcot in his observations on hysterical hemianæsthesia.

Varieties of cutaneous anæsthesia in hysteria.

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|---|---|---|
| (1) Total affecting all forms of sensation. | { | (a.) Complete—anæsthesia proper. |
| | | (b.) Incomplete—hypæsthesia. |
| (2) Partial, affecting certain forms of sensation only. | { | (a.) Loss of sensation to pain only—analgesia. |
| | | (b.) Thermo-anæsthesia, loss of sensation to temperature but not to touch or pain. |
| | | (c.) Loss to touch and pain with preservation of temperature sense. |
| | | (d.) Loss of sensation to electrical stimulation only. |
| | | (e.) Preservation of sensation to electrical stimulation alone with loss of other forms of sensation. |

In testing for sensory defects, an assistant, with his finger and thumb, closes the eyelids of the patient, this procedure avoids all source of error from the slipping of a bandage. For tactile sensibility it is sufficient to touch the skin with the index-finger, by using greater or less force, the complete or incomplete loss of tactile sensation is judged. The patient counts aloud 1, 2, 3, 4, &c., as soon as he perceives the contact, and thus an idea can be formed if perception is normal or retarded. Objects of different form or weight are placed in his hands, in order to test his perception of form and weight. M. Charcot always employs only the simplest procedures, and never uses complicated æsthesiometers. It is necessary to carry out the investigation quickly as otherwise one may sometimes provoke the phenomena of transference. To test for analgesia a pin, with a fine sharp point is used; it is necessary to make the pricks sufficiently profound, and to go over an extensive surface of the skin, or at any rate to prick the skin in several places widely separated from each other. The results may be then, for convenience, marked out on a chart.

Pitres has shown that the nerve-trunks are insensible. He calls attention to the theoretical interest of these observations. "They prove, unless I am deceived, that hysterical anæsthesia does not result from a functional inertia of the terminal extremities of the sensory nerves. For if the seat of this anæsthesia were peripheral, the direct irritation of the nerve-trunks should arouse sen-

sations of pain in the nerve-centres which remain intact. Since it is possible to strike and prick the nerves of the limbs without provoking any sensory reaction, the seat of the anæsthesia must be above the nerves, and as there are only the nerve-centres above them, the determining cause of hysterical anæsthesias must be sought in these centres."

In the healthy, a violent blow on the epigastrium is sometimes immediately fatal, a lighter one gives rise to a passing syncope, this effect M. Pitres has never observed to be produced in an hysteric. He gives an example where forcible blows on the epigastrium of a patient caused her to feel a sensation of well-being, and she smiled at blows which would have made a strong man faint. He considers that the abdominal and thoracic viscera are anæsthetic in hysteria. The bones, ligaments, and articulations are also frequently insensitive.

For the investigation of thermo-anæsthesia, M. Charcot uses a thermometer with a flat bulb, the lower part of the tube and the bulb being enclosed in two metallic cylinders. The outer cylinder glides over the other, and can be raised in order to see that the apparatus is in good order. The inner cylinder is filled with copper filings. The outer cylinder is warmed in the flame of a spirit lamp, care being taken that the heat is not too suddenly applied, and that the index of the thermometer does not pass beyond 100° C. The position of the index being observed, and the base of the outer cylinder applied to the skin, accurate observations on the temperature-sense can be made.

As a general rule, anæsthesia involves all forms of sensation in the affected areas, but the different forms are frequently dissociated, one of the most important varieties being the simulation of the affection of sensation met with in syringomyelia. M. Charcot found that of seventeen cases, in eleven the anæsthesia was of the common type, in two, sensibility to touch and pain was preserved, but the temperature-sense was lost, and in four, the condition corresponded exactly to that described in cases of syringomyelia; *i.e.*, tactile sensation was normal, whilst sensibility to pain, heat, and cold, was either absolutely lost or very greatly deficient. Isolated loss of the tactile sense does not seem to occur.

Distribution of cutaneous anæsthesia.—General anæsthesia of the skin is rare as a lasting symptom, (one per cent. of cases): as a temporary symptom it is more common, often appearing for some hours after a convulsive seizure; for instance, it occurred after a convulsion in a patient generally hemi-anæsthetic, and

then gave place to hemi-anæsthesia again. In such cases there is abolition of the special senses, complete amaurosis, deafness, and absolute loss of taste and smell.

In the most common form, the distribution of the anæsthesia is unilateral. In some cases the hemi-anæsthesia is rigorously unilateral, in others, the sensory disturbance is not uniform over the affected side.

Cutaneous hemi-anæsthesia is accompanied with sensory disturbances of the mucous membranes, special senses and deep tissues, *predominating* on the side of the cutaneous affection; predominating but not rigorously limited to this side. M. Gilles de la Tourette has never seen a case, and he quotes some recent observations of Thomsen and Oppenheim to the same effect, in which the skin, mucous membranes, and special sense organs, were simultaneously affected on one side of the body, whilst on the opposite side they remained absolutely normal. When the field of vision is contracted on the anæsthetic it is always a little contracted on the other side also. Hearing is often equally affected or equally spared on both sides. Speaking generally, the parts of the mucous membranes which border on the skin, are similarly affected to it. On the other hand, the mucous membrane of the larynx, of the epiglottis, of the posterior part of the nasal fossæ, of the tympanum and the integument of the bony portion of the auditory canal, preserve some independence relatively to the cutaneous anæsthesia, and are completely anæsthetic, hypo-æsthetic, or normally sensitive over their whole extent. This point is of practical importance, for according to Lori and Ott, anæsthesia of the deeper mucous membranes, especially of the larynx and epiglottis, is always exactly unilateral in cases of hemi-anæsthesia due to an organic intra-cerebral lesion.

(c) The anæsthesia may be distributed in disseminated islets, of which it is impossible, from their variability, to give a methodical description. The anæsthetic patches occur indifferently on the head, trunk, or limbs. Their number and size are very variable. Their form does not correspond to the anatomical distribution of the sensory nerves, nor to that of the blood-vessels of the skin. More or less extensive anæsthesia of the mucous membranes, and of the special senses is concomitant, and there is generally some loss of muscular power on the side on which the loss of sensation predominates.

Hysterical anæsthesia is often found in clearly circumscribed areas, the knowledge of which we owe to M. Charcot, who showed that, in male hysteria, in certain cases of brachial mono-

plegia anæsthesia was superimposed on the paralysis, and in such a way, that at that time, when syringomyelia was still imperfectly known, its distribution alone was sufficient for diagnosis. In a case of paralysis of one upper extremity, the shape of the anæsthetic area recalled one of those pieces of sixteenth-century armour designed to protect the shoulder—and also affected almost the whole arm. In paralysis of the shoulder only, the anæsthesia is limited below by a horizontal plane perpendicular to the axis of the limb, situated at two fingers' breadths above the elbow in front, and posteriorly passing above the olecranon. In paralysis limited to the elbow, the lower line of the anæsthesia is circular and horizontal, situated about two fingers' breadths above the wrist, in a plane perpendicular to the longitudinal axis of the limb. M. Charcot concludes that "this arrangement in *geometrical segments* delimited by circular lines in planes perpendicular to the long axis of the limb, represent, at least for the limbs, the type of anæsthesias of cortical origin, whatever the lesion may be. Only in hysteria this characteristic will generally be much more marked and easy to recognize than in organic focal lesions . . ." (49) It is unnecessary to add that the lower extremities may be affected in the same way as the upper. Hysterical anæsthesia does not follow the distribution of the cutaneous sensory nerves.

Anæsthesia may appear suddenly, may undergo many sudden modifications, or may spontaneously disappear: its extent and severity in any particular case are often in correspondence with the severity and extent of the other symptoms. It is valuable in diagnosis, because of the impossibility of its being successfully simulated.

The principal symptoms associated with hysterical cutaneous anæsthesia are: (i.) abolition of the superficial reflexes. M. Pitres has seen the abdominal reflex preserved, though feeble, on the anæsthetic side in cases of hemi-anæsthesia, he has also seen it absent on both sides in hysteria with diminished sensibility. Both he and M. Gilles de la Tourette agree "that there is no reason to attribute to this symptom the diagnostic value which Rosenbach's observations seemed to give it." This author stated (50) that the abdominal reflex is lost in certain cases of cerebral lesions, but is always preserved without alteration in hysteria.

(ii.) Preservation of the organic reflexes, *e.g.*, reddening of the skin by a mustard leaf, or the raising of a blister by a vesicatory. The pupil also dilates on sensory stimulation to a sharp pinch or

prick, even of the anæsthetic part of the skin. Rosenberg's observation, that in cutaneous anæsthesia the insensitive parts readily take the temperature of the surrounding medium, does not hold good for hysteria.

(iii.) Very remarkable as contrasted with anæsthesia of organic origin, is the absence of disagreeable subjective sensations, or of notable disability to perform the common actions of every-day life.

M. Pitres says that "he has closely questioned the hysterical patients under his observation and he can affirm that they were for the most part unconscious of their sensory troubles (anæsthesia). When pressed some of them said that they had remarked that they did not feel any pain when accidentally pricked or injured in particular parts. Others alleged that the anæsthetic limbs were less adroit at work, which demanded precision of movement. One hemianæsthetic patient said that she perceived the temperature of the water of a bath on one side of the body only. None of them suffered from the presence of uncomfortable or painful sensations, (numbness, tingling, prickings, pins and needles, &c.) in the anæsthetic areas of the skin. This is an important peculiarity, for generally anæsthesias dependent on organic lesions of the spinal cord or peripheral nerves are accompanied by subjective sensations and functional troubles which strongly attract the attention both of the patient and of the doctor. Hysterical anæsthesia, on the contrary, is a latent symptom that the physician must seek out by a regular and methodical exploration."

M. Gilles de la Tourette agrees with Prof. Pitres as to this, "although he has sometimes noted subjective sensations of numbness, and formication, and sometimes more or less acute pain in the anæsthetic limbs. A patient with total general anæsthesia avoided, without knowing the reason, walking in the dark. The difficulty she found was explained by the fact that when she closed her eyes she could no longer stand because she did not feel the contact of the ground. A strong and vigorous man walked about the courts of the Salpêtrière during very cold weather in his shirt sleeves without appearing to feel cold. This singularity gave him no uneasiness and he was altogether unaware of his thermo-anæsthesia."

Hysterical patients with hemianæsthesia complain most frequently of feeling the whole side of the body heavy; the arm of this side is more awkward and weaker than ordinarily. "With some reservations we do not hesitate to conclude, that in the

great majority of cases anæsthesia requires to be sought for and is almost always first recognized at the medical investigation.'

Professor Pitres does not agree with Briquet's statements (*op. cit.* p. 283) that "the temperature of the anæsthetic skin is lowered one or two degrees Centigrade," and the circulation through the capillaries is slowed. On the contrary, he finds that neither the circulation nor the temperature is altered on the anæsthetic side. Sweating is as easily induced by pilocarpine, the skin is reddened by irritants, becomes inflamed and suppurates exactly in the same way in the anæsthetic as in the sensible parts. He concludes that there is no reason to believe that hysterical anæsthesia depends on a local disturbance of the circulation.

It appears to be certain however that there is a real ischemia in the anæsthetic regions, shown by the fact that the anæsthetic skin rarely bleeds in the site of pricks. M. Charcot relates some instances of this kind (51). "Some leeches having been applied to a patient with hysterical hemianæsthesia, I remarked that the bites bled with great difficulty on the anæsthetic side, whilst on the healthy side, there was the usual amount of bleeding. Grisolle, a very skilled and exact observer, noticed the same thing. This ischæmia, which by the way very rarely attains to this degree, explains certain reputed miraculous circumstances. For example, in the epidemic of St. Médard, it is said that sword-cuts did not give rise to hæmorrhage in those affected with convulsions."

M. Pitres thinks that the contact of a foreign body excites a strong contraction of the cutaneous vessels before the blood has time to flow. When the anæsthetic skin is pricked, a pale areola, soon surrounded by a wider zone of redness, appears around the pin-prick, and quickly gives place to a raised papule or minute wheal, which persists for half an hour to an hour or longer. After the papule has formed, a little serous fluid may flow from the orifice of the prick. There is thus produced an immediate contraction of the vessels in the site of the puncture, and a localized œdema limited to this spot.

He goes on to say that this "vascular hyper-excitability" which prevents the flow of blood after a prick, is sometimes general, and not limited to the anæsthetic region.

If, however, a blister, which always reddens the skin over the insensible areas, be first applied, pricks over the reddened surface bleed freely, though sensation often remains unaffected.

In commenting on these observations, M. Gilles de la Tourette states that in a series of experiments which he made with M. Cathelineau on ten hysterical patients, for the purpose of

studying the composition of the blood in hysteria, they never noticed that the wet-cupplings bled differently on the anæsthetic and on the sensitive side of the body. The simple preparatory application of the cupping glass did not play in this case the same rôle as the blister described above, for it reddened equally the skin of each side. (52)

M. Guichon has further shown (53) in opposition to some observations of M. Teissier of Lyons, that subcutaneous absorption takes place as well on the anæsthetic as on the sound side.

M. Pitres has found in some cases a form of paræsthesia in which the contact with the skin of substances, which commonly only cause a sense of contact, gives rise to intensely painful sensations: he gives the name of haphalgesia to this symptom. He also relates two cases in one of which if a piece of copper, brass, gold, or silver were placed in the hand on the anæsthetic side of the body, a strong contraction of the limbs resulted, the patient became agitated and after a few seconds was convulsed. In the other contact with silver caused a burning sensation, which rapidly increased until it became so acute as to cause loud cries or even a convulsive attack. Other substances were inert. The experiments made were so carefully guarded that intentional deception would seem to have been impossible. M. Gilles de la Tourette has seen one such case only: this patient could not bear the contact of gold, and could not wear ornaments of this metal on account of the burning sensation produced by them.

Anæsthesia, though very frequent, may be absent in cases of confirmed hysteria. M. Charcot teaches that in children especially hysteria presents itself in a monosymptomatic form, paralysis, contracture, rhythmical chorea, or other symptom without any affection of general or special sensation. Of 40 adult patients M. Pitres found two free from any sensory defect.

Out of 235 patients Briquet observed general anæsthesia in 4, hemi-anæsthesia in 93, and anæsthesia in patches in 138. Hemi-anæsthesia occurs more often on the left than on the right side.

In the muscles there is loss of pain on pressure. The muscle, which is insensitive to pressure between the fingers, is readily excitable on percussion or electrical stimulation, in other words, in spite of its analgesia, its contractile power remains intact. There is loss of the muscular sense and therefore some uncertainty of voluntary movement with the eyes closed, and of the ability to distinguish between different weights; there is also loss of the sense of fatigue which normally follows on sustained muscular effort.

The loss of the sense of muscular contraction may be a cause of awkwardness or uncertainty of movement but not of paralysis. In other cases again the patients can perform any movement with their eyes open, but when the eyes are closed they are unable to accomplish any action. Lasègue (54) showed in 1864 that if the sense of touch were utilized, *e.g.*, by placing the anæsthetic hand on the unaffected skin of the head, movements of the hand could be carried out with the eyes closed, so that touch could thus be substituted for sight. Professor Pitres in a case where the left arm was anæsthetic, found that the grasp of the left hand was nil with the eyes closed and 50 kilos with the eyes open. If his hand was placed—the eyes being shut—on the table and he was told to move his fingers as if playing the piano, he could not do so, but if the hand was placed on the thigh he moved the fingers at once. He shows also that regular or rhythmical movements if begun with the eyes open can be continued after they are closed, but if stopped cannot be initiated anew without opening the eyes, and that bilaterally associated movements can be started and carried out with closed eyelids, *e.g.*, the patient mentioned above could perfectly perform the movements of the arms for swimming when he was entirely unable to move the left arm alone without the aid of sight. He considers that the phenomena described by Duchenne under the name of paralysis of the muscular sense depend chiefly on a partial disturbance of motor innervation. This disturbance, of central origin, can be corrected by certain sensory impressions and by several modes of bringing into action the motor centres. It is not simply an affection of the muscular sensibility. Anæsthesia of the muscles may be one of its constituent elements but its real origin is outside the muscles, and even outside the centres of sensory perception.

The sense of position of the limbs may be preserved in the absence of muscular sensation, of the sense of fatigue, and of that of contraction of the muscles; these senses do not therefore depend the one on the other. As a general rule however, the sense of position is perverted or abolished when there is profound anæsthesia of the muscles and skin. Not only the sense of position of the limbs but that of the size and weight of objects handled may be lost. If there is general anæsthesia the patient may be lifted out of his bed on to the ground, if his eyes be closed, without his perceiving the change in his position. If he is suddenly ordered, whilst walking, to close his eyes, he will fall heavily to the ground.

Normally we are conscious of the existence of the several parts of our body, this consciousness being the sum total of the numer-

ous sensory impressions of all kinds which reach the brain from all parts. It is a curious fact that hysterical patients, in spite of complete anæsthesia of the skin and deeper tissues, often retain the notion of the existence of their members. Only in certain cases of both motor and sensory paralysis is the sense of the existence of the limbs absent. M. Pitres thinks that further observations are required to determine whether the cause of this loss lies in the nerves or in the sensory centres.

Hysterical anæsthesia of the mucous membranes and of the special senses.—The most common forms are total anæsthesia, analgesia or hypoæsthesia; haphalgnesia may also be observed. The patients are generally as unconscious of anæsthesia of the mucous membranes as of that of the skin when they first come under observation. The “organic reflexes” are preserved; an irritant placed in contact with the tongue, conjunctiva, or nasal mucous membrane provokes a flow of saliva, tears, or nasal mucus respectively. These affections of the mucous membranes may be the first or the only hysterical stigmata to appear. When cutaneous anæsthesia is general, the mucous membranes and special senses are affected on both sides: when it is unilateral, on that side only.

The loss of the special sense is added to that of the general sensibility of the part; that is to say, in the majority of cases the anæsthesia affects not only the special sense organ itself, but also the mucous membrane or skin which covers it.

This rule especially holds in hemianæsthesia. There, as M. Charcot has shown, the special senses are abolished or obscured on the anæsthetic side, and as a rule on that side only. The eye is an exception, for generally the concentric contraction of the visual field is bilateral. At the same time it is always greater on the anæsthetic side. In these cases also the insensibility of the buccal mucous membrane may be complete on both sides.

Anæsthesia of the anus and of the urinary passages is, according to Briquet, very rare, much more so than that of the mucous membrane of the genital organs. In the vagina the anæsthesia is often limited to one side of it.

Unilateral loss of taste is common, and may exist as an isolated “stigma.” M. Lichtwitz (55) has found in some cases that together with partial loss of taste on parts normally gustatory, taste was present on other parts of the buccal mucous membrane, *e.g.*, the palatine vault, which have, as a rule, no such function.

He used the galvanic current as the test in his researches and

states: (i. that in six cases of hysteria the "field of taste" (gustatory area) was diminished or abolished; (ii.) the gustatory area determined in this way was either of the same extent or smaller than that for sapid substances; (iii.) there seemed to be no relation between taste and general sensation as determined by galvanism.

M. Pitres divides the patients from this point of view into four classes:—

(i.) Those in whom taste and common sensation are lost together over certain areas of the tongue.

(ii.) In others taste is lost whilst common sensation remains intact.

(iii.) Sometimes taste is preserved over parts that are insensible to pain and temperature.

(iv.) In some there are areas of tactile anæsthesia over the tongue, and of loss of taste, the limits of which do not correspond.

M. Charcot and M. Gilles de la Tourette have found, however, that in the great majority of a very large number of cases examined by them, loss of common sensation and of the special sense are superimposed upon one another, not only in the case of taste but of the other special senses.

The comparative frequency of affection of taste in hysteria, as compared with other nervous diseases, renders it a valuable symptom in diagnosis.

The frequency of anæsthesia of the larynx is very variable in hysteria; according to Thaon (56) one-sixth of hysterical patients are affected with it. It affects both sides of the larynx, and is never unilateral: it is often associated with paralysis of the vocal cords.

As regards the nasal mucous membrane, M. Lichtwitz (57) says, that it is, perhaps, of all the mucous membranes, the least affected with anæsthesia. His conclusions are opposed to those of Briquet, which have met with general acceptance. Briquet wrote: "The circumference of the anterior orifice of the nasal fossæ, and all parts of these cavities which are accessible to instruments, may be pricked with a pin, without the patient feeling it." At the same time, M. Lichtwitz has very frequently observed the presence of spasmogenic or hyperæsthetic areas in the nasal fossæ. It is difficult to see why the nasal mucous membrane should be exempted from the general rule that M. Charcot teaches, namely, that the mucous membranes participate in the anæsthesia of the affected side in hysterical hemianæsthesia, the affection of the special senses being superimposed on that of common sensation.

In eleven cases, Lichtwitz found unilateral anosmia in four, total anosmia in one, bilateral loss of smell for certain odours only in one, and once a pronounced diminution of smell in the left nostril only. Several patients found asafœtida not disagreeable—one was hyper-sensitive to all odours.

Hysterical deafness may be absolute; coming on suddenly it may last for days or weeks, and as suddenly disappear, but in most cases it is partial only. Generally this deafness is associated with loss of common sensation over the external auditory meatus and tympanic membrane, but this is not always the case.

The reader is referred to Walton's observations on deafness in hysterical hemianæsthesia in *BRAIN*, vol. i., pp. 458-472, 1883.

The deaf ear is often affected with tinnitus. Sometimes the sounds in the ear are so loud and persistent as to resemble those of Ménière's disease; and then, should an attack with vertiginous symptoms come on, the difficulty of diagnosis is great. In these cases, the auditory meatus or the auricle, is probably the seat of an hysterogenic zone.

It is not doubtful, M. Gilles de la Tourette remarks, that the shocks sustained by the organism in general, whether the patient be previously exempt from the neurosis or not, have a special influence on the genesis of anæsthesias of the skin or special senses, whether these are extensive, total, in patches, or as is generally the case, unilateral. Local injuries have a direct action in the production of geometrically-distributed cutaneous anæsthesias. M. Charcot relates the case of a woman, who after giving a box on the ear to her child, was taken directly after with hysterical paralysis of the hand, with the addition of anæsthesia. In another case, the anæsthesia, which affected the paraplegic lower limbs, was exactly limited by a circular line at the level of the imaginary passage over the body of the wheels of a carriage which had knocked the patient down. (58). In such cases, the topographical distribution of the anæsthesia seems to be determined by the paralysis. Very often the anæsthesia persists after the cure of the paralysis, an important element in the prognosis, for so long as the anæsthesia remains, the paralysis may return.

As regards the mucous membranes, and especially those which cover the special sense organs, in the majority of cases, anæsthesia supervenes without any recognizable direct cause, but in a fair number of cases, a relation between the exciting cause and the anæsthesia can be traced. For instance, a blow from the fist, or with a whip, may directly give rise to amblyopia. Note, however,

that local causes (traumatisms) seem to play no part in the genesis of gustatory troubles. The influence of all causes is limited to determining the localization of sensory symptoms on the common ground-work of the neurosis.

The data as to the mode of onset of hysterical anæsthesia are incomplete, but it may probably be either sudden or gradual. Anæsthesia may replace hyperæsthesia or *vice versâ*. We have fuller knowledge as to the course of hemianæsthesia.

It is one of the most fixed (permanent) of hysterical stigmata, and this applies especially to hemianæsthesia with the corresponding defect of the special senses. In an hysterical woman, æt. 65, at the Salpêtrière, hemianæsthesia still persists after thirty years, though all other signs of the neurosis have gone; and in another, in whom the diagnosis was confirmed at the autopsy, it existed for more than forty years.

With regard to the occurrence of anæsthesia, cutaneous and of the special senses, at different ages, M. Charcot teaches that it belongs especially to the adult age, both in men and women. In the old, the symptoms of hysteria gradually die away, but anæsthesia may persist as the two cases just quoted show. On the other hand, in children sensory troubles are more rare. They may be present, but never to any great extent. Some disturbance of taste, of hearing, a slight retraction of the visual field, are the forms most often met with in them. In accordance with this the hysteria of children is less severe and is capable in a large number of cases of absolute cure, whilst on the other hand it is especially obstinate and refractory to treatment in the adult. Hence the importance with regard to prognosis of the sensory stigmata; their presence indicates a severe type of the disease.

Briquet and Duchenne found that anæsthesia could be caused to disappear by faradisation.

The researches of MM. Charcot and Burq showed that by the contact of certain metals, or of a magnet, by the use of electrical currents, or of mechanical vibrations, the sensibility of the anæsthetic parts could be restored. They found an idiosyncrasy to the action of certain metals in particular patients; that in one anæsthesia disappeared under the influence of iron or copper, and in another under that of gold or zinc. At the same time the phenomenon of "transference" was discovered, that is to say the anæsthesia under the action of the "æsthesiogenic agent" left the side of the body first affected and passed to the other one. Subsequently many other substances were found to act in the same

way. M. Pitres shows by the aid of experiments with mercury that it is not necessary for the metals to be in a solid state. Mercurial vapour and liquid mercury were equally efficacious. Of the four gases, hydrogen, coal gas, oxygen, and carbonic acid, the former only was effectual.

The following short account of the phenomena of metalloscopy is by M. P. Richer (59). "Suppose a patient to be affected with hemianæsthesia. Several metals are applied to the cutaneous surface without affecting the anæsthesia; at last that one is applied—gold for instance—to which the patient is sensitive. Two or three twenty-franc pieces—one is sufficient—are placed on some part of the anæsthetic side of the body, say on the forearm. In a few minutes the patient feels a sensation of heaviness and tingling in the limb. If then, the eyes being closed, he is pricked with a pin in the immediate neighbourhood of the coin, he feels pain; if he is pricked at a distance of some centimetres from it the pricks are no more felt than before. A little later this area of sensibility extends, and in extreme cases it may happen that the anæsthetic side wholly recovers its sensation momentarily. We say momentarily, because if the experiment is prolonged the anæsthesia re-appears, beginning in those parts in which it disappeared last, and the patient returns to his former state. The total duration including the disappearance and re-establishment of the anæsthesia, varies with the surface-area affected, lasting, from half a minute to three-quarters of an hour or more."

Accompanying the return of sensation is an increased supply of blood: pricks cause bleeding now whereas they were before bloodless, there is a rise of temperature in the part, and increased muscular power.

Vice versâ by applying the metal to the sound side of a hemianæsthetic patient, the anæsthesia may be caused to transfer itself to this side. If the experiment is prolonged however, the side first affected again becomes anæsthetic.

Consecutive oscillations. Three varieties of consecutive oscillations are described by P. Richer:—

- (i.) The patient wholly recovers sensibility for a time.
- (ii.) He remains in a state of "transference," *i.e.*, if he had left hemianæsthesia before the experiment, he has, for some instants, right hemianæsthesia.
- (iii.) Finally he relapses into the state prior to the experiment.

M. Pitres thinks that it is probable that "transference" is simply an exaggeration of a well known physiological phenomenon. In 1854 Hoppe (60) observed that if the thumb and index finger

of one hand are sharply pricked there is a marked diminution of sensibility on the symmetrical points of the other hand. Other authors have made similar observations, notably, those cases in which transference of the motor areas of Jacksonian epilepsy by the application of a circular blister above the point of origin of the primitive area has occurred. These observations show that transference is not such a strange phenomenon as it appears at first sight, but is found in an attenuated form under normal and pathological conditions altogether independent of hysteria.

It is certain that the æsthesiogenic agents, metallic or non-metallic, do not produce constant and uniform effects on all subjects. Every patient is influenced by such or such an agent to the exclusion of others, and nothing in the character of the anæsthesia enables us to foretell what means will be effectual. Moreover the patients may vary in their idiosyncrasy to the influence of different agents from one moment to another.

(1) Two different metals applied simultaneously can reciprocally annihilate each other's influence. Vigouroux (61) says, "If in an hemianæsthetic patient, in whom the application of gold causes the anæsthesia rapidly to disappear, a piece of gold is applied, and above it a piece of silver, so long as they remain in contact the anæsthesia is not modified. If the silver is removed the anæsthesia disappears. If when sensation has been recovered, one does no more, it soon disappears and anæsthesia returns. But if the piece of silver is replaced, the sensibility remains so long as the silver is left there. The action of the silver has been in each case to render permanent the condition of things existing at the time it was applied." In an hemianæsthetic patient the phenomenon of transference, effected by an active metal, can be made to persist indefinitely by the application of a neutral one to the side formerly sensitive.

(2) When hysterical patients are improving, but have not regained complete health, before the anæsthesia completely disappears, it is in a sort of unstable equilibrium. If in this state an active metal is applied the anæsthesia reappears in all its original severity.

Theories advanced to explain these phenomena :—

(1) Physical theories.—The committee of the Société de Biologie with the aid of M. Regnard, came to the following conclusions (i.) Metals applied to the human skin determine currents of a sufficient intensity to be measured by a very delicate galvanometer ; (ii.) the intensity of the current varies with the different metals and with the state of the skin ; (iii.) the passage through the

skin of a galvanic current, of an equal intensity to the current determined in it by the application of an active metal, produces exactly the same effects on sensation as the metal itself.

They found further that a feeble current (*a*), or a strong current (*c*), caused a rapid return of sensibility while one of intermediate strength (*b*) had no effect. The difference in the action of the metals may be explained by supposing that in a certain patient gold produced a current of equal intensity to (*a*), iron to (*b*) and copper to (*c*); without invoking the aid of any mysterious agencies, gold and copper would then be active, and iron inert.

(*b*) On the other hand M. Vigouroux thinks that "the action of the metals is a phenomenon of static electricity." He brings forward several experiments in support of this, especially the fact of the neutralisation of the æsthesiogenic action of two metals by the superposition of one upon the other, which can hardly be explained on the supposition of electrical currents. Briefly he holds (1) that a certain electrical state of the peripheral organs of general and special sense is necessary for the performance of their functions. (2) This electrical state can be modified and with it the sensibility by producing on the surface of the body a change of tension, and this can be done by the application of a polarised plaque, by electrification with one pole alone or by the galvanic current.

(*c*) Professor Schiff for several reasons does not believe in these electrical theories. He thinks with the physicists that material bodies are the seat of rapid molecular vibrations which they can communicate to neighbouring bodies. The variety of action of the metals will depend, according to him, on the number and rhythm of the vibrations of the æsthesiogenic agent, and of their concordance or otherwise with those of the nervous system of the subject.

According to another theory, supported by Hughes Bennett and Hack Tuke, the æsthesiogenic influence of these substances is to be referred to psychical causes, and is chiefly brought about by the imagination.

M. Pitres thinks that the phenomena of æsthesiogenesis do not all depend upon the same cause. The sensibility can be modified both by psychical and by physical influences. This we should expect, for there are many physiological and pathological phenomena which undergo variations dependent both upon external and upon psychical excitations. The proofs which demonstrate the intervention of physical causes in the production of certain of the æsthesiogenic phenomena are numerous and convincing.

On the other hand, the powerful æsthesiogenic influences of certain psychical factors—imagination, expectancy, moral emotions, and verbal suggestions is beyond question.

From the characters of hysterical anæsthesia, M. Pitres thinks that the localisation of the disorder of the nervous centres is in the basal ganglia, and that it is due to a functional inertia of these bodies. Anatomo-clinical observations show that destructive lesions, limited to the sensory region of the bulb or of the cerebral peduncles, give rise to anæsthesias that present all the clinical features of those met with in hysteria, except their variability. Experimental ablation of the higher parts of the brain affords support to the view, that the bulb may be considered as the nervous centre “for the change of impressions into sensations.” From the fact that organic lesions of the posterior part of the internal capsule produce hemianæsthesia of the opposite side of the body, it has been held that hysterical hemianæsthesia depends upon a functional defect of these fibres. He adduces several observations in which, after hemiplegia of organic origin, there has been hemianæsthesia, which has undergone “transference,” or has disappeared under the action of æsthesiogenic agents, and he thinks that this hemianæsthesia is not to be attributed to disease of the internal capsule, but to the indirect effects of the lesion in inhibiting the grey matter of the subjacent basal centres.

On the ground of the clinical differences between anæsthesias of cortical origin in organic disease and hysterical anæsthesias, Prof. Pitres cannot accept M. Binet's conclusions, based on experiments on hysterical subjects, that the lesion in hysteria, of whatever nature it may be, has its seat in the cortex.

As to diagnosis, three morbid conditions are considered to give rise to hemianæsthesia, with corresponding unilateral affection of the special sense organs; (1) a lesion of the posterior part of the internal capsule; (2) the intoxications, especially alcoholism and plumbism; and (3) hysteria. When such symptoms occur in the second group of cases, M. Charcot has shown that the patients are affected with hysteria, in addition to lead or alcohol (62). He also states, (63) “that hemianæsthesia due to organic lesion of the internal capsule, shows no essential difference from that of hysteria.” The chief aid in diagnosis is that when, as is the rule, hemianæsthesia from an organic lesion is accompanied with hemiplegia, the lower third of the face is paralysed, whilst in hysterical hemiplegia, the face almost invariably escapes. Until 1889, M. Charcot's researches led him to believe that the anæs-

thesias disposed in geometrical areas, altogether independent of the nerve supply of the part—"en manchon, en gigot, en brassard" were typical of hysteria, but since that time observations on syringomyelia have shown that in this disease, the anæsthetic areas may have a similar arrangement. Moreover, the peculiar dissociation of the different forms of sensation characteristic of syringomyelia may be simulated by hysteria.

Hysterical Amblyopia and Achromatopsia.—Briquet observed that when amaurosis is unilateral, the patients are only occasionally aware of it.

Anæsthesia more commonly affects the eyes than the other special sense organs. Insensibility of the conjunctivæ, especially of the left conjunctiva, is so constant that it may be regarded as a characteristic sign of hysteria. When anæsthesia is complete, the oculo-palpebral reflex is absent. The loss of sensibility is superimposed on amblyopia. M. Féré (64) says: "The hysterical patients with hemianæsthesia that we have observed, and in whom there was neither contraction of the visual field, nor achromatopsia, preserved the sensibility of the conjunctivæ: those who have loss of vision for one or several colours, and have a more or less regularly proportioned contraction of the visual field, have lost the conjunctival sensibility, and those who have complete loss of colour vision, and almost complete loss of the visual field (for white), have lost sensibility over both conjunctiva and cornea."

Contact with the conjunctiva produces a flow of tears, even in cases where the oculo-palpebral reflex is lost.

The field of vision in hysteria is concentrically contracted. It is well-known that in the course of one examination, the field of vision in hysterical patients may be frequently modified, if the examination be too protracted, or so long continued as to fatigue the patient. The investigation must, therefore, be rapidly made, the determination of the field in its vertical and horizontal diameters being in most cases sufficient.

The contraction of the visual field is generally bilateral, but greater on one side, that of the hemianæsthesia, than on the other. Occasionally it is unilateral when it is on the side of the hemianæsthesia.

When the macula is invaded there is complete amaurosis. Unilateral is, relatively to bilateral amaurosis, the more frequent; the latter seems to be very rare. In the cases that M. Gilles de la Tourette has seen, the blindness was only

temporary in the great majority: it most frequently appeared after a convulsive attack, disappeared as suddenly as it came on, lasting some hours, or at most a few days. In one case, however, reported by Harlan (65) blindness persisted for ten years.

In hysteria, the different circles which correspond in exploration to the limits of the field of vision for each colour are, more or less, concentrically contracted, following the order recognised in the normal state. Vision for violet, green, red, and orange may disappear in the order named, these being the colours which are normally perceived centrally; the fields for the two first being the most restricted. Yellow and blue, the fields which have the greatest peripheral extent, disappear the last. Vision for these two colours is, therefore, longest preserved in hysterical amblyopia. But there are exceptions to this rule, red and not blue being frequently the last to go. In a still higher degree of hysterical achromatopsia the form of objects is perceived, but they all appear grey.

These disturbances of colour-sense are most marked in the eye on the anæsthetic side of the body, but the other eye generally shows them in less degree.

Permanent dyschromatopsia may suddenly give place to complete achromatopsia, under the influence of an impending convulsive attack; and this may disappear as suddenly after the paroxysm is over.

Central scotoma is rarely observed in hysteria. M. Parinaud (66) has found that central scotoma and peripheral contraction of the field of vision may develop simultaneously, so that perception of colours may be lost at the centre and at the periphery of the retina, and be retained in an intermediate zone which forms a ring surrounding the fixation point. Another peculiarity is that when anæsthesia to light and the contraction of the field for white are the most marked features, the field for red may be more extensive than that for white.

Acuteness of vision is as a rule very little modified in hysterical amblyopia, thus affording a remarkable contrast with the amblyopias that occur in atrophies of the optic disc.

Hysterical amblyopia alters very little the perception of form, but is often accompanied by other troubles of accommodation or of refraction such as the polyopia or diplopia that has been so well described by M. Parinaud (67). This polyopia is often overlooked, because it is generally obscured by amblyopia, and is only noticed by the patient when methodically sought for. M. Parinaud gives the following rules for its detection (68). "A

pencil or some similar object is held before the eye and slowly removed. At first it is seen single; at 10—15cm. distance from the eye, a second image appears, generally on the temporal side. As the object is removed, the two images separate, and not seldom a third appears, less distinct, and on the opposite side. If the object is still further removed, the images become indistinct. At the same time the presence of macropsia or micropsia is shown, by the removal or approximation of the object."

No ophthalmoscopic changes have been found in cases of hysterical amblyopia.

M. Parinaud has observed that a certain number of patients cannot distinguish colours with either eye alone, and yet distinguish them very well in binocular vision. Also that in many cases of unilateral concentric contraction of the visual field, it is sufficient to open the sound eye for the field of vision of the amblyopic eye to increase from 10 to 20 degrees.

Monocular amblyopia, and even binocular retraction of the visual field does not inconvenience the patient much, so long as the acuteness of vision and perception of form is not modified. Similarly for achromatopsia; but one must bear in mind that railway servants who have developed achromatopsia as one of the symptoms of hysteria evoked by a collision may become incapable of carrying on their daily work.

How are we then to explain the fact that hysterical patients seem to suffer no inconvenience from their amblyopia in ordinary binocular vision? They cannot voluntarily neglect the image received by the affected eye, as persons who squint do, for they do not fix an object with the sound eye only, but place it exactly in the (principal) axis of binocular vision. M. Pitres believes that the amblyopia is corrected, because it exists only in monocular vision and disappears at once when both eyes are used. He tested a patient, who had barely perception of light with the left eye, with Flees' box, an apparatus in which two points of different colours are seen, one to the right and the other to the left, and by an ingenious arrangement the point to the left is seen with the right eye and *vice versa*; he also employed the prism and the screen tests. To all these tests she answered correctly, showing apparently that binocular vision was normal. He quotes some observations of M. Parinaud, who found that certain patients had achromatopsia for each eye in monocular vision, and yet distinguished colours perfectly in binocular vision. Two explanations of these facts have been proposed, the first, sup-

ported by Ferrier's experiments and which the author considers most probably correct, that the cortical centres for binocular and monocular vision are distinct, and that the latter only are affected in hysterical amblyopia. The second, advanced by M. Bernheim, that hysterical amblyopia (*cécité*) is a purely psychical phenomenon, an illusion of the mind. "The hysterical person," he says, "neutralises unconsciously by the aid of his imagination, the visual image that is normally perceived." The author forcibly refutes the argument that hysterical amblyopic affections are simulated.

Causes of amblyopia.—The onset is generally insidious, but a local traumatism (to the eye) may according to the law established by Charcot, develop local hysterical phenomena. The prognosis is good.

Differential diagnosis.—Lesions of the posterior third of the internal capsule, according to M. Charcot, are accompanied by hemianæsthesia with concentric contraction of the visual field and dichromatopsia identical with that observed in hysteria; this contraction of the visual field is bilateral, and M. Charcot has therefore rejected the term crossed amblyopia by which he at first designated this affection.

The amblyopia of lead-poisoning belongs in reality to hysteria, as do many cases of alcoholic amblyopia also. Cases of genuine alcoholic amblyopia also occur, and are distinguished by the presence of a central scotoma spreading to the periphery (69). It is true that central scotoma may occasionally occur in hysteria, and a case of combined hysteria and alcoholism has been reported by M. Charcot (70) in which there was both concentric contraction of the field (hysteria) and central scotoma (alcohol), but a knowledge of these variations will be sufficient to prevent mistakes.

Disseminated sclerosis is frequently associated with hysteria, and syphilitic lesions have been found in the fundus of an eye affected with hysterical amaurosis. MM. Déjérine and Tuitant (71) have stated that in syringomyelia a concentric contraction of the visual field for colours is found, but only corresponds to that of hysteria in the field for white. For other colours the visual field is irregular, notched, and shows the greatest contraction for green; all these features being unlike the phenomena of hysteria, and more resembling those found in atrophy of the optic disc.

Ophthalmoscopic examination showed no lesion in these cases. As no other examples of the same kind have been reported, whilst many cases of syringomyelia have been published, in which, apart from a morbid association of the disease with hysteria, the visual

fields have been normal, the author concludes with M. Brianceau (72) that—

(i.) In the majority of cases of syringomyelia the visual field remains normal: contraction of it is not a symptom of the disease.

(ii.) When it is retracted an explanation other than the cord lesion is to be sought for.

(iii.) Hysteria, so often associated with syringomyelia, is the only cause of contraction of the visual field in this disease apart from local ocular affections.

M. Féré (73) states that all the manifestations of epilepsy attended with loss of consciousness are followed by amblyopia for a certain time: he always found a decided contraction of the visual field (18 patients), when he examined the patients within half-an-hour after consciousness had been regained. This contraction is accompanied by a diminution of the acuteness of vision by two, three, or four-fifths. The sensibility to light and to colours is also lessened (after the fits) and this diminution seems to be the counterpart of the hyper-sensibility to light and to colours—particularly red (Hammond)—that is sometimes observed in the prodromal period. Further M. Féré has three times found anæsthesia of the eyelids, conjunctiva, and the circumferential skin around the eye coincidently with complex visual troubles and nystagmus. He does not admit the existence of permanent contraction of the field of vision except in cases where the attacks closely follow each other. MM. Oppenheim and Thomson on the other hand, in 94 cases found in only 4 temporary, while one-third of the cases showed permanent contraction of the visual field (74). M. Pichon (75) similarly found a permanent contraction of the field of vision in one-third of his patients, but he insists on its irregularity of contour. M. Hitier however, investigating the question anew on 87 epileptics in the service of M. Charcot, found a permanent contraction in 3 only. M. Charcot teaches that permanent contraction of the visual field in epilepsy is “une quantité négligeable,” above all when it is compared with the temporary contraction that follows the fits. In the great majority of cases he says some contraction follows the fits, but it may occasionally precede them as the aura. When however the fits follow each other at short intervals, every 6 or 8 days for instance, the contraction of the visual field is to some extent a permanent symptom.

Hysterical affections of the ocular muscles.—These affections in the great majority of cases accompany amblyopia, and often for a short time relatively to the persistence of amblyopia in hysterical patients. Sometimes—and this is especially true of one

variety, blepharospasm—they exist alone, as the sole symptoms of an “abortive” hysteria, and the most frequent instances of this, so far as regards the localization of the symptoms in the eyes, are found in children. Most often however they are accompanied with other well-marked signs of hysteria.

There may be loss of the muscular sense in the orbital muscles. Szokalski (76) says, “If the anæsthesia of the ocular muscles remains free from all complication with retinal anæsthesia, the faculty of vision is disturbed by loss of the muscular sense in these muscles, or loss of the sense of position of objects seen. The patient seems to lose the perception of height and depth, of what is vertical and what is horizontal, &c.”

M. Borel (77) has undertaken some experiments to elucidate this question. The subjects were a man and a woman suffering from total anæsthesia, with absolute loss of the muscular sense in all four limbs. The man (who fell like a log as soon as his eyelids were closed), with one eye shut was made to look through a tube so directed as not to permit of his seeing any object which was familiar to him and could help him. The outlook from the tube was closed at some distance by the hand. One tried to make him follow with the eye alterations in the axis of the tube, and after noting the position of the eye, he was questioned as to its direction. He always gave wrong answers, when, indeed, he did not indicate that he had no idea of the direction of his eyes. He did not know whether he was looking up or down, to the right or to the left. It sufficed for the tube to be well fitted to the globe of the eye so that no familiar object could be seen to put him completely at fault. The same procedure gave identical results in the other patient. Loss of muscular sense may accordingly be as complete in the muscles of the eye as in those of the limbs.

Monocular polyopia and micro-megalopsia appear to be caused by a contracture of the ciliary muscle. M. Parinaud says that the ciliary muscle is very often affected in hysteria. Its contraction generally accompanies that of other muscles of the eyeball and of the orbicularis, but may exist alone. It is often overlooked as it is masked by the amblyopia present. This contraction of the muscle of accommodation produces effects analogous to those of myopia, that is to say the p.r. is more or less approximated. But hysterical contracture differs from myopia in that the muscle being immobilised in the position of contraction, the range of accommodation is nil, the p.r. and p.p. are the same, and the eye is adapted for one distance only. This contraction may be

extreme and the point of distinct vision be very near the eye. In myopia the same degree of contraction will bring this point nearer, in hypermetropia place it further away. In the last case the eye being adapted for a considerable distance convex glasses will correct the vision for near objects, *e.g.*, for reading. In cases of this kind the condition may be mistaken for paralysis of accommodation which is a very rare phenomenon in hysteria. M. Parinaud explains monocular polyopia in the following way. The crystalline lens is not completely homogeneous in structure. The segmentation of its substance together with the modifications of curvature that it undergoes in accommodation render it comparable to a lens divided into several segments, which are re-united, but not fused together, in such a way that the images formed by each of the segments are combined at the common focus but remain distinct on the near and far side of it. The normal action of accommodation, which is to bring the rays of light always to a focus on the retina, hides this defect of structure, but it becomes manifest in certain troubles of accommodation. In this way atropine produces for certain distances monocular polyopia. It fixes the ciliary muscle and brings out especially in young subjects the segmentary state of the lens whilst at the same time exaggerating its curvature. M. Parinaud attributes micro-megalopsia to a similar contraction of the muscle of accommodation. It is more frequent than monocular polyopia, which arises from the same cause, and which requires for its production, a stronger contraction and certain peculiar conditions.

Blepharospasm is the most frequent contraction of the ocular muscles. It may occur in hysterical subjects from very slight causes. Numerous instances have been reported which place the influence of hysterogenic or hyperæsthetic zones on the production of blepharospasm beyond dispute. Hysterical blepharospasm may be clonic, tonic, or take the form that has been called by M. Parinaud "pseudo-paralytic ptosis." The clonic form is the most common and also the most transitory. It is always bilateral. Tonic blepharospasm occurs in two forms, painful and not painful. In the former it is as a rule bilateral, and the muscular contraction is so forcible that it can only be overcome with difficulty or not at all. There is generally photo-phobia and lachrymation. In the latter the affection is often unilateral. The orbicularis is actively contracted, the upper eyelid thrown into folds, and the patient's efforts to open the eye only result in feeble contractions of the muscle, which accentuate the closure of the lids, whilst the upper eyelid shows from time to time convulsive movements or flick-

erings. When one tries to raise the eyelid a very appreciable resistance is felt. In the pseudo-paralytic form, the tonic spasm is so to speak absent; when the patient is told to open the eye he instinctively throws the head backwards and the frontalis contracts energetically, as in true paralytic ptosis. But the upper lid covers the lower exactly, when it is raised with the finger it descends again more energetically than in paralysis, and the convulsive flickerings of the upper lid may be seen in this form also. M. Charcot has recently (78) pointed out that the eyebrow is lowered in spasm whilst in paralysis it lies higher than that of the healthy side. Lastly M. Gilles de la Tourette has discovered a symptom which he regards as pathognomonic, and which consists in the addition of sensory troubles to the spasm. In the painless form of blepharospasm there is a zone of anæsthesia circular in shape covering the eye and the surrounding skin for a distance of 2cm. beyond the orbit. The conjunctiva, palpebral and ocular, is almost completely insensible. On the cornea sensation is preserved over its internal segment only.

In painful blepharospasm there is the same circular zone, but in this case of hyperæsthesia, instead of anæsthesia. These phenomena are analogous to the sensory troubles "in geometrical-territories" superimposed on loss of function, and independent of the distribution of the peripheral nerves, that MM. Charcot and Féré had already noticed in cases of hysterical amblyopia.

Blepharospasm is almost always associated with amblyopia. It is generally of short duration, but may last some years; like other hysterical phenomena it may disappear suddenly. The surgical procedures which have been advocated, namely, stretching, excision of the nerve, or section of the orbicularis muscle are quite unjustifiable.

M. Parinaud (79) states that tonic hysterical blepharospasm is almost always accompanied by contraction of some of the ocular muscles, but that isolated contraction, at least a state of fixed contraction, is rare in these muscles, though a passing diplopia is common enough. Spasmodic strabismus in hysteria has resulted from the presence of carious teeth or alveolar periostitis. During the convulsive attack, at the period of the tonic or clonic convulsions, contractions of the ocular muscles may be noted, and they may sometimes coincide with contractions of the neck muscles, so that conjugate deviation of the eyes and head may be simulated, and suggest an organic cerebral lesion. Generally, however, the above syndrome is not so accentuated, the eye is carried in all directions by rapid movements of short duration without

any regularity ; at the same time the pupils are agitated by spasmodic contractions and dilatations. Permanent deviation of the eyes is much more rare. A case is recorded by Frost (80), in which there was conjugate deviation of the eyes downwards and to the right. The author considers that in almost all, if not all, the reported cases of hysterical paralysis of the third nerve, the accuracy of the diagnosis may be doubted. He cannot at all accept Schäfer's conclusions (81) on hysteria in children, that whilst paralysis of the facial and hypo-glossal nerves is rare, that of the third nerve, or isolated paralysis of the branch to the levator palpebræ is sufficiently common. The only case of hysterical paralysis of the third nerve that is above dispute, is one communicated by Parinaud and P. Richer, and even that dates from ten years ago, when the diagnosis between spasm and paralysis was not so well differentiated as it is to-day. In this case there was paralytic ptosis, paralysis of accommodation, and incomplete palsy of the right internal and inferior rectus.

Permanent dilatation or contraction of the pupils is very rare in hysteria, and is generally observed as a transitory phenomenon of the convulsive attacks. In some cases dilatation of the pupil has been noted following myosis due to spasmodic contraction of several days' duration, and may be compared to the weakness of a limb which follows a state of hysterical contraction. Spasmodic mydriasis has been described by Giraud Teulon (82) and Dubois (83).

Insufficiency of convergence, from weakness of the internal recti, is so bound up with other ocular motor troubles, that it will only very rarely be met with as an isolated symptom.

As to the question of hysterical paralysis of the sixth nerve, M. G. de la Tourette quotes three cases only, one of which was a case of associated paralysis of the sixth pair of nerves in looking to the left or to the right ; in the two others the paralysis at first affected the external rectus of one eye, and then passed to the same muscle of the other eye. This alternation of the paralysis appears to be typical of hysteria, the paralysis of organic origin being much more fixed, and the secondary spasm never showing the same degree of intensity.

Cases of external ophthalmoplegia in hysteria have been reported by Ballet (84), Bristowe (85), Parinaud (86), and Raymond (87). These observations show that external ophthalmoplegia in hysteria has some peculiar characters which differentiate it from the same symptom when due to an organic lesion. There is an entire absence of all voluntary movement, while unconscious (in-

voluntary) movements are preserved : “the eyes are not constantly fixed, but execute movements of limited extent in different directions.” There is no mydriasis; further the ophthalmoplegia remains external, and is not internal or total.

Hysterical hyperæsthesia.—Hyperæsthesia may affect the skin, mucous membranes, or viscera.

In searching for hyperæsthesia, it is important to examine a sufficiently extensive surface at a time. Further, stroking the skin with the finger will be much more vividly felt than a prick, especially a deep prick, for the skin itself and not the underlying tissues is the seat of the pain. Cutaneous hyperæsthesia is especially found in the hysteria of adults, male and female. It appears and disappears spontaneously. Convulsive attacks have a marked influence, hyperæsthesia appears or disappears after them, or is replaced by anæsthesia, and *vice versâ*.

In distribution, hyperæsthesia is rarely general. M. Gilles de la Tourette has observed a few cases of this in men. Most commonly it is disposed in patches. Unlike anæsthesia there is no need to search for hyperæsthesia, the patient is acutely conscious of it, partly from exaggeration of subjective sensations (numbness, formication, prickings, tinglings), and partly from exaggeration of sensations excited directly by external agencies (blows, rubbing, &c.).

Occasionally hyperæsthesia is unilateral. In one case described by M. Charcot, there was hyperæsthesia on one side of the body, whilst the other side was anæsthetic. Sometimes hyperæsthesia occurs in geometrical areas; it is then superimposed on the loss of function of the part, *e.g.*, in a case of contraction of the orbicularis palpebrarum, there was a zone of hyperæsthesia exactly overlying this muscle. Hyperæsthesia probably appears first, and always persists a certain time after the contraction has gone.

Hysterical Joint Affections.—According to M. Charcot, the knee is the joint most often affected. Of seventy cases the knee was affected thirty-eight times, the hip eighteen times, the wrist eight, the shoulder four, and the ankle twice.

Causes (1) Injury, a fall, blow, or unaccustomed fatigue, such as a prolonged march. M. Charcot has shown that to have their due effect, these traumatisms, which are often insignificant in themselves, must take place in persons whose mental state is peculiar, such as hysterical persons who at times are as open to suggestion as those who are in the hypnotic state. There is a “traumatic suggestion” rather than an actual traumatism, for

there is always, between the reception of the injury and the manifestation of the hysterical symptom, a period of preparation, "*méditation psychique*." Imitation, as Paget has shown, sometimes plays an important part. The age of the patients varies. Men are frequently affected. In opposition to Briquet M. Charcot teaches that an hysterical joint may be one of the first manifestations of the neurosis.

The first and earliest symptom of an hysterical joint is pain, the second, spasmodic contraction of the muscles which put the joint into action. This contraction may spread to all the muscles of the limb. The third is loss of power in the limb. Brodie's classical description of the symptoms is quoted, in which it is pointed out that the seat of the pain is in the skin. In the case of the hip the area of cutaneous hyperæsthesia is roughly triangular, the apex of the triangle being at the root of the scrotum, or mons Veneris, and the base running parallel to the fold of the groin, the triangle enlarges progressively as it turns round the buttock to pass towards the sacrum and encroaches on the hypogastric region. In the knee the hyperæsthesia extends round the limb a hand's breadth above and below the middle of the joint, it has a similar distribution for the ankle and the joints of the upper extremity. This hyperæsthesia is added to the loss of function of the joint, and its distribution may be compared with that of the anæsthesia which accompanies spasmodic contractions of the orbicularis palpebrarum, or of the limb muscles. These hyperæsthesias or anæsthesias of geometrical distribution appear before the loss of function and persist after it. Sometimes these hyperæsthetic areas become true hysterogenic zones.

Charcot (88) and Babinsky (89) have shown that muscular atrophy analogous to that described by Valtat (90) and accompanied by the reaction of degeneration and fibrillar trembling may be present in cases of hysteria arthralgia. This muscular atrophy is less precisely localized than in cases of organic disease, in which it always affects the extensors of the joint: moreover it is much more rare. Examination of the joint under chloroform should be practised. M. Charcot (91) has shown that the order in which the painful phenomena return with the regain of consciousness after chloroform, is of diagnostic importance. He says "During the return to consciousness, rigidity began to reappear to a certain degree in the affected muscles before any manifestation of pain in the joint occurred. The sensibility of the skin had already partly returned, the patient had begun to reply to some questions, at a time when the sensibility of the

deeper parts (percussion of the trochanter or heel) was still in no way exaggerated."

In a case of organic hip-disease pain returns first in the deeper, afterwards in the superficial parts. The changes that have been found in the joints and bones in long-standing hysterical cases can be accounted for by the want of use of the parts. The duration of hysterical arthralgia may be months or years, the general course being interrupted by temporary recoveries, to be again followed by a relapse.

M. Charcot has shown that when anæsthesia or hyperæsthesia of geometrical distribution exists in addition to a contraction or paralysis with or without arthralgia, the sensory disturbance always persists some time after the disappearance of the functional troubles. So long as the sensory disturbance persists one ought to fear the return of the paralysis, contraction, or arthralgia. Like all other hysterical affections arthralgia may undergo a sudden cure.

As a rule, the cause of the appearance of hyperæsthetic zones remains unfathomable. In a case reported by M. Charcot, however, a man fell into the water whilst fishing, and the cord of the net became entangled round his left leg. He was nearly drowned, and a patch of hyperæsthesia was afterwards found over the spot compressed by the cord. M. Pitres also relates the case of a patient "who imagined that she was, from time to time, visited by an old woman who touched her in different parts of the body, and on all the spots thus touched, there was afterwards hyperalgesia which persisted for some days."

Hysterogenic zones.—Hysterogenic zones are circumscribed regions of the body, which may or may not be painful, from which there often pass, during the prodromal stage of the spontaneous attacks, special sensations which play a part in the whole of the phenomena of the hysterical aura. Compression of these spots has for its effect, either to determine a convulsive seizure, or a part of the spasmodic phenomena of the seizure, or to arrest suddenly convulsions already begun. This compression not only arrests the convulsive attacks, but also attacks of rhythmical chorea, of cough, hiccough, &c., in short, every kind of paroxysm. Hysterogenic zones are excitant (spasmogènes) or inhibitory, (frénatrices) of convulsive attacks, some, especially those which are not the seat of hyperæsthesia, are "spasmo-frénatrices," that is to say, light pressure may provoke and firm pressure arrest an attack. These zones are especially found in hysteria with convulsions. They are often

of small dimensions, of the size of a two or five franc piece. The skin over them is generally anæsthetic, a fact which differentiates them from areas of simple hyperæsthesia, it may, however, be hyperæsthetic. When seated on the hemianæsthetic side of a patient they may easily be overlooked. Firm pressure is necessary for their discovery. The occurrence of spontaneous pains may be a useful guide to the position of hysterogenic zones. They appear and disappear most frequently under the influence of an hysterical fit.

MM. Bourneville, Regnard, and P. Richer state that hysterogenic zones occur only on the head and trunk, but M. Pitres has also found them on the limbs. According to him, the zones on the limbs and thorax are exclusively "excitant" (spasmogènes), and those over the ovarian and epigastric regions, "inhibitory" (frénatrices).

He classifies hysterogenic zones according to their anatomical distribution into—

(1) Cutaneous, which are the least common.

(2) Subcutaneous, which are more frequently met with. In this variety, convulsive attacks are determined by pressure on subcutaneous tissues, irritation of the skin, or of the underlying muscles and tendons being without effect. He thinks that the seat of the irritation is in the nerves, and that the nerve tracks leading to the zone, if compressed in any part of their course between the spinal cord and the zone, can equally well excite an attack. This statement applies to the subcutaneous zones only.

(3) Visceral zones are situated in the parenchyma of complex organs, such as the mammary gland, ovary, and testicle. In such cases, the skin overlying the affected organ has no hysterogenic properties.

On some special hyperæsthetic hysterogenic zones.—Those on the scalp are some of the most important and may be single or multiple; when single, they are generally situated at the vertex. They are of limited extent and the sensibility over them is exquisitely acute; spontaneous radiations of pain start from them. Hysterical cephalalgia is found in six-sevenths of hysterical female patients according to Briquet, but though often caused by the presence of an hyperæsthetic or hysterogenic zone on the scalp, this is not always the case. Briquet concludes his description of "clavus" thus. "The pain of clavus is fixed and does not move about. It lasts several days or even three or four weeks. It is frequently accompanied by attacks of shivering, vomiting,

digestive disturbance and sometimes by fever." This observation contains the germ of the description of a recently studied group of symptoms, namely, hysterical pseudo-meningitis. In this affection the symptoms of meningitis are very closely simulated. It generally occurs in adult women but has been once observed in a little girl of five (92). The mode of onset may be sudden or preceded by a prodromal period of malaise as in tubercular meningitis. The chief symptom in the latter case is headache which shows itself in the form of acute exacerbations and coincides with the presence of hysterogenic or hyperæsthetic zones on the scalp, which should always be sought for. These exacerbations are accompanied by cries and groans. Sometimes there is delirium. There is often photophobia and diplopia from passing strabismus: there may be rigidity of the neck muscles, opisthotonus, and contractions of the arms or of the legs. The pulse may be slowed, but according to Pitres is never irregular. Hyperæsthesia and *tâche cérébrale* are present, and constipation and vomiting complete the list of symptoms, which thus closely resembles those of tubercular meningitis. In one case only was the temperature raised to 39.2° (102.5° F.) in the others it did not exceed 38.6° (101.4° F.) and in several remained normal. Meanwhile the evolution of the symptoms is not continuous but proceeds by exacerbations or paroxysms. The presence of some concurrent and independent acute inflammatory affection must be looked for, since this may be the cause of the fever in cases where pyrexia is present; for instance, vaginitis was found in two cases. M. Chantemesse (93) says that the mode of onset and the progressive evolution of the symptoms cause them closely to resemble tubercular meningitis. Only the knowledge of the patient's antecedents, of the presence of sensory troubles attributable to hysteria and the normal temperature should make one suspect hysteria. He (94) has also made use of the observations of MM. Gilles de la Tourette and Cathelineau on the changes in the urine in hysterical paroxysms. They showed that during hysterical paroxysms the amount of urea excreted is diminished and the proportion of the earthy to the alkaline phosphates, which is 1-3, both in the healthy and in the hysterical subject apart from the paroxysms, becomes 1 to 2 or equal. M. Chantemesse finds that these changes are found in the urine of persons suffering from hysterical pseudo-meningitis, and this disorder must therefore be probably regarded as an attack or crisis spread over a long period.

M. Gilles de la Tourette has observed five cases of facial

neuralgia of hysterical origin. In one patient in Prof. Charcot's clinic, there had occurred three attacks of facial neuralgia at different times. An hysterogenic zone was found at the point of exit of the nerves, and another of the same kind in the intercostal region. Pressure on the zones excited an attack of neuralgia ending in a convulsive attack. These neuralgias are to be considered as a painful prolonged crisis when they end in a fit of convulsions, and as a complete hysterical paroxysm when no convulsions occur. The radiation of the pains is interesting: it not rarely occurs between neighbouring hysterogenic zones; for instance the pains may start from the vertex and arouse hysterogenic zones of the fifth pair, or acting at a still greater distance compression of an ovarian zone may excite an attack of hysterical migraine. As to diagnosis from true facial neuralgia M. Charcot lays stress on the occurrence of the hysterical affection in the evening, whilst the genuine attack generally takes place in the morning. Most important is the co-existence of other hysterical stigmata. The author thinks that when the nature of a facial neuralgia is uncertain, an estimation of the urinary solids during the period of 24 hours in which the attack occurs should be made and compared with that of another period of 24 hours free from the paroxysm.

Rachialgia is one of the most common hysterical manifestations.

The hyperæsthetic hysterogenic area may extend the whole length of the spinal column, more often it stretches only over four or five vertebræ, generally in the dorsal and lumbar region. It is particularly well-marked over the spines and does not pass laterally beyond the spinal furrow. It is tender to the slightest touch and is generally the seat of spontaneous pain. Pressure upon the zone may determine a convulsive attack. The observations of Brodie on rachialgia are complete and well known. It should be noted that M. Paul Richer (95) has pointed out that the lumbar spines are, in some persons, especially when they bend forward, very prominent, and that this normal prominence has been frequently treated as pathological.

Sometimes rachialgia is accompanied by a lateral hyperæsthetic or hysterogenous zone on the trunk,—pleuralgia of Briquet—it extends round one side of the body from the spinal column behind to the middle line in front, and is generally from 4-5 fingers' breadths in width and seated about the level of the 5th, 6th, 7th and 8th intercostal spaces. The pain is aggravated by breathing, coughing, and walking.

The knowledge of the occurrence of this hyperæsthetic zone is important, for with a painful area over the dorsal spine, spontaneous pains, and sensation of constriction round the waist, the girdle-pains and exquisite sensibility of the thorax and spine found in some cases of locomotor ataxy may be closely simulated. The hyperæsthetic hysterogenic zone of the vertebral column may be localised in its inferior extremity when it constitutes hysterical sacrodynia and is a very obstinate affection. Its nature can be recognised by the co-existence of other hysterical stigmata, and by the extreme sensibility of the overlying skin, in which the pain is chiefly localised.

In men two hysterogenic cutaneous zones are often found. One pseudo-ovarian is seated in the region which corresponds to the ovary in the woman. It is generally unilateral and on the anæsthetic or hyperæsthetic side of the body, but may be bilateral. Its extent is limited and the skin over it is often insensible, deep pressure being necessary to induce an attack. The second is seated in the skin of the scrotum (not in the testicle itself), and it is almost always unilateral.

Hysterogenic zones on the mucous membranes.—Our knowledge of these zones we owe largely to the observations of a pupil of Professor Pitres, M. Lichtwitz. M. Lichtwitz has found only spasmogenic zones, apart from the "hypnogenic" zones to be mentioned later. They are situated on the mucous membrane of the nose, mouth, upper air passages, external auditory meatus and lachrymal canal. They frequently co-exist with cutaneous hysterogenic zones in the same subject. He found that they occurred more particularly on the anæsthetic side of the body, a statement which M. Gilles de la Tourette thinks requires further investigation. Unlike the skin, which shows no naked-eye changes over the position of a zone, obvious morbid changes are frequently present in the affected mucous membranes: *e.g.*, inflammatory affections of the larynx and external auditory meatus, atrophy or hypertrophy of the mucosa over the lower turbinated bones. Beyond direct pressure these zones may be excited by faradic and galvanic currents, by chemical substances, and by inhalations. It is important to note that in some cases the cure of the local morbid changes in the mucous membrane has been followed by disappearance of the hysterogenic zones and of the hysterical symptoms. The special senses differ in the way in which they react to the various hysterical excitations. When an hysterogenic zone is situated on the lingual or nasal mucous membrane the trouble excited by it remains as a rule local, as a perversion of

smell. When the mucous membrane of the tympanum, middle ear or eye is implicated, more complex phenomena follow the irritation of the zone. The excitation of hysterogenic zones localised in certain regions may give rise to a symptom-complex generally observed under very different circumstances, for instance facial neuralgia, when the aura is seated on the branches of the fifth. In the case of the ear the effects influence the nerves of the labyrinth and convulsive attacks arising from a zone in this situation may exactly simulate Menière's disease. The diagnosis may be made by the characteristic changes in the relative proportion of the urinary solids already referred to as occurring during an hysterical convulsion.

Hysterogenic zones may be seated on the conjunctiva, cornea, or lachrymal duct, and irritation of them may give rise to a convulsive attack, in which all the symptoms of migraine may be simulated. The chief points in the diagnosis of the hysterical form are the following. Migraine may constitute the whole of an hysterical paroxysm, or be the starting point of a convulsive attack. Besides the ordinary prodromata of an hysterical attack, throbbing in the temples, globus, &c., there is almost always an hysterogenic zone which forms the starting point of the paroxysm; this zone may be seated in the eye itself, or on the vertex. In one case the pain began at the vertex, passed to the left supra-orbital region, and was followed by a luminous scotoma. In two other cases, pressure on an hysterogenic zone in one over the sixth dorsal vertebra, and in the other over the left ovary immediately caused the appearance of the scintillating scotoma (96). When migraine forms the aura of an attack, or when it constitutes the whole (specialised) paroxysm and starts from an hysterogenic zone, irritation of which determines the attack, the presence of the hysterical form of migraine is certain. In doubtful cases the author relies on an examination of the urine (see below). Hemipopia is often observed in ordinary migraine, and may be present in the hysterical form, as a temporary phenomenon of the attack, but is never permanent. In these latter cases there is the usual concentric contraction of the visual field, and M. Parinaud, examining an hysterical patient in whom an attack of migraine with hemipopia could be induced by hypnotic suggestion, found that, during the presence of hemipopia, the visual field was still more contracted than in the usual state, but the objective characters of permanent hemipopia were not present.

Vaginismus is most often the result of a spasmodic contraction of the vaginal muscles brought about by irritation of an hyperæsthetic or hysterogenic zone.

The viscera most frequently affected are the breast, the testicle, and the ovary. In the first two the zone is generally unilateral, and may be seated either only in the skin covering the testicle or breast, or in the glands themselves. Professor Pitres says that the painful affection described by surgeons under the name of irritable testicle, is sometimes the consequence of a symptomatic neuralgia of the spermatic plexus, but in a large number of cases appears to be due to a simple hysterical hyperalgesia of the parenchyma of the testicle. Castration has been practised, but the pains have often persisted after removal of the testicles, proving that the hyperalgesia is not always due to local irritation of the nerves of the spermatic plexus, and that it depends, at least sometimes, on a functional modification of the sensory nervous centres.

Hyperæsthetic hysterogenic ovarian zone.—The efficacy of pressure on the abdomen in arresting hysterical convulsive seizures was known to Mercado in the sixteenth century; Schutzemberger exactly localised the spot in 1844; he says that the ovary forms the most frequent point of departure of an hysterical spasm. Briquet pursuing his general theory as to the seat of hysterical hyperæsthesia localised ovarian pain in the abdominal walls; he gave it the name of cœlialgia.

An ovarian zone rarely passes unnoticed for it is often the point of departure of particular sensations which attract the patient's attention. Ordinarily there is between the attacks a dull pain, brought out by walking or by the pressure of a corset. For some hours or days before a convulsive seizure, the pain becomes sharp, lancinating, and radiates into the epigastric region, to be followed by globus, in a word the common sensations of the classical aura happen. After the attack is over the pain becomes less acute. Sometimes it is sufficiently severe to cause the patients to walk bent forwards and inclined to one side in order to relax the abdominal muscles.

In some cases the skin is hyperæsthetic in a limited area over the site of the ovary. The author believes that these cases have been examined shortly after convulsive attacks, when the skin is itself the seat of an hyperæsthetic zone, for in the great majority there is on the contrary a very circumscribed zone of cutaneous anæsthesia lying above the ovary. According to M. Charcot's writings, by deep pressure at the intersection of the line joining the anterior superior iliac crests and that limiting the epigastric region laterally, the brim of the pelvis may be felt, and at about the middle of this rigid crest a small ovoid body will be felt to

glide under the fingers. Pressure on this brings out not ordinary sensations of pain but those of the hysterical aura. In two cases where the patients were pregnant, this ovarian zone was observed to rise with the normal ascent of the ovary during pregnancy. M. Guinon reports an additional instance in point. This zone is generally unilateral, it is one of the most fixed, active, and powerful zones; it generally has two properties, light pressure upon it exciting, strong pressure arresting, an hysterical attack.

Hysterogenic zones can be made to disappear by (1) general and (2) local means.

(1) Includes static electricity, the galvanic current applied to nervous centres, ether or chloroform anæsthesia.

(2) Artificial local anæmia, by Esmarch's elastic band round a limb, application of cold (ether spray), a mixture of ice and salt, of a mustard poultice, hypodermic injections of pure water, localised electrification. The action of any of these measures is only temporary.

M. Pitres says that "in localised electrification" of the zones, the current must be differently applied, according as subcutaneous or cutaneous zones are in question. In the first case, the electrodes may be applied directly over the skin of the hysterogenic region. In the second, the zone itself must be avoided. The electrodes must be placed beyond the limits of the excitable region. Further, the currents employed must not be too strong, or they may produce convulsions before the zone has lost its excitability.

The prodromal period of a spontaneous hysterical seizure is marked in its complete form by three stages. (1) The psychological aura, marked by ideas of sadness, terror, &c.; (2) hysterogenic aura, acute pains at situation of the hysterogenic zones; (3) ovarian aura, the sensation of a ball which rises upwards from the ovarian regions, and immediately precedes the onset of convulsions. A convulsive attack may be artificially provoked by giving rise to the sensations of either of these three periods. M. Pitres thinks that possibly the clumsy handling of a patient in an hysterical seizure may give rise to irritation of hysterogenic zones and so prolong the attack. The knowledge of the presence of these zones is important to the physician for (1) he will take care that hysterical convulsions are not prolonged in the above way. (2) He will be able to artificially provoke an attack by their means, with the object of putting an end to long-continued painful auras, or of modifying certain permanent hysterical symptoms, *e.g.*, contractures, or of confirming a doubtful diagnosis. (3) He will be

furnished with a very efficacious means, in many cases, of arresting the attacks.

The provocation of a paroxysm may be as useful as its arrest in modifying the manifestations of hysteria actually present. For instance an hysterical paraplegia may be brought to an end by inducing a convulsive attack by the aid of an hysterogenic zone. The paraplegia may indeed be replaced by a spasmodic contraction or some other hysterical phenomenon, but an hysterical manifestation of recent date is always more easily relieved than one of long standing. The displacement of an hysterical phenomenon is already a step towards its disappearance. Certain hysterical states, *e.g.*, hysterical sleep which may last for days or weeks, may often be suddenly brought to an end by pressure on an hysterogenic zone.

Hysterical or pseudo-angina pectoris.—Hysterical angina, unlike the true form, occurs especially in those under 40. Whenever an attack has occurred, it is probable that other paroxysms in the same patient will take this form. Hysterical angina is a rare affection. The attacks are generally sudden and often nocturnal, sometimes they recur with a regular periodicity. There may be an hyperæsthetic or hysterogenic zone in the præcordal region, or over the mid-dorsal region of the spine. The attack begins by an increase of pain and tenderness in these zones. Sometimes the aura may begin with pains in the testicles or abdomen. The pains radiate to the neck and down the left arm to the fingers, or they may start from the fingers and pass to the chest. This is the neuralgic form. In the “vaso-motor” form the face becomes pale and cold, then livid, red and purplish, and the same changes may affect the left arm. The state of the heart, pulse, and respiration show the greatest differences in different cases. The pupils may be dilated or contracted. The patient may experience a feeling of inexpressible terror, or may have a tendency to syncope. A kind of dream of the attack may pass through all this period. In the diagnosis from true angina pectoris the exciting cause is important. Moral agencies, such as a contrariety, bring on the hysterical, while physical causes induce the organic form. The pulse and respiration generally remain calm in true angina—the cardiac erethism so especially related to the cries, groans, and agitation of the hysterical crisis does not occur in them.

The number of attacks is uncertain. One patient had more than two hundred in the course of two years. In diagnosis, the family and antecedent history of the patients, must be care-

fully enquired into, and the presence of hysterical stigmata sought for; the analysis of the urine on the lines mentioned above, will be of service; organic disease of the heart and great vessels must be excluded, the symptomatology of the attacks studied, and by this means an accurate conclusion can generally be arrived at.

The convulsive seizures of hysteria.—Hysterical convulsions are more common in men than in women, of M. Pitres' cases, 56 out of 61 women, whilst only 7 out of 31 men, had convulsions. A strong emotion is most often the cause of the first attack, afterwards they may arise spontaneously, or be excited by the most trivial causes.

To M. Charcot, and after him to Paul Richer, the credit is due of being the first to give a systematic description of hysterical convulsive attacks and to show that they may be divided into several phases, succeeding each other in regular order. The "grande attaque" of hystéro-épilepsie is composed of the following episodes:—

I.—Premonitory symptoms. Auras.

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| II.—Stages of the attack proper. | { | a. Epileptic period. | { | (1) Phase of tonic contraction. |
| | | | | (2) Phase of clonic contraction. |
| | | | | (3) Phase of resolution. |
| | | b. Period of contortions and of "clownism." | { | (1) Phase of illogical attitudes. |
| | | | | (2) Phase of "grands mouvements." |
| | | c. Period of passionate attitudes. | | |
| | | d. Period of delirium. | | |

III.—Terminal phenomena. Contractions, paralyses, &c.

M. Pitres remarks that all these phenomena are not seen in the attacks of ordinary hysteria. True as this description is in the cases from the study of which it was drawn up, there are marked differences between the attacks of common or "minor" hysteria, and of hystero-epilepsy or severer hysteria (*la grande hystérie*). The two affections belong to the same natural family of disease, but are not identical in the details of their manifestations. The convulsions of common hysteria are less complete and less regular. Certain phases of the hystero-epileptic attack are wanting. The epileptoid phase is very short, that of the passionate attitudes is absent, and the period of delirium, when present, ought to be placed rather amongst the closing

phenomena than in the active period of the attack. He thinks it useless to complicate the description of ordinary hysterical convulsions by seeking to identify them with those of hystero-epilepsy, and divides them into three stages only: (1) the pre-convulsive stage; (2) the stage of convulsions; (3) the post-convulsive stage.

- I.—Pre-convulsive period. {
1. Stage of psychical auras: the patients are anxious, restless, sad; they laugh or cry.
 2. Stage of sensory auras: pains in various parts of the body, most intense in hysterogenic zones.
 3. Stage of abdominal aura: a feeling of a ball, which rises from the ovarian, or sometimes from the epigastric region to the throat, causing a feeling of suffocation.

- II.—Convulsive period. {
1. Phase of tonic contraction: generally very short, the limbs becoming rigid for some seconds.
 2. Phase of clonic convulsions: begins by one or several loud screams, followed by rapid, purposeless movements of different kinds; opisthotonus is frequent; intervals of arrest of convulsions, followed by fresh ones; the whole lasting 15 to 30 minutes or longer. From time to time the features are contorted into various expressions. Rarely the patients try to get up and escape from those who hold them.

- III.—Post-convulsive period. {
- The patients remain for a certain time after the convulsions have ceased with eyes closed and limbs flaccid. After some minutes a talkative delirium begins. They may in this be either unconscious, or may reply momentarily to questions, lapsing again into delirium. This stage may be less obvious, the patient merely sobbing or laughing for a short time.

Finally, the delirium ceases, the patients appear for a moment astonished, and ask what has happened: after a short attack they resume their occupation, after a longer one they remain in a state of lassitude which makes them repose for some hours. They never pass into the state of stertorous sleep which succeeds an epileptic seizure.

The author divides hysterical seizures into (1) incomplete, in which one or other of the usual stages is absent; and (2) abnormal, in which the convulsive phenomena, attenuated or

modified in their symptoms, present very different characters from those commonly present in the regular attacks.

There are three varieties of "incomplete" convulsions; in the first, the pre-convulsive period, in the second, the post-convulsive, and in the third, both the pre- and the post-convulsive stages, are absent. The first variety is rare.

The abnormal attacks present the most varied symptoms, and sometimes closely resemble epileptic convulsions.

M. Pitres enumerates the distinctive features of the past history, symptoms of attacks, and general examination of the patients in the intervals, by which the differential diagnosis between epileptic and hysterical convulsions may be made. In doubtful cases, the examination of the urine passed in the twenty-four hours succeeding a fit, may give valuable information. After an epileptic fit, all the urinary constituents, particularly the urea, and phosphates are increased in quantity, and the proportion of the earthy to the alkaline phosphates, 1 to 3, remains normal; whilst after an hysterical convulsion the total amount of urea and phosphates is diminished, and the quantity of the earthy and alkaline phosphates excreted tends to be equal.

Contraction diathesis—"diathèse de contracture."—Of this disturbance of the motor functions to which M. Charcot gave the name of diathesis, or state of liability to contraction, M. Richer says (97) "This state is neither paralysis nor contraction and yet partakes of the nature of both. Its presence is not betrayed by any objective sign: the patient preserves all his freedom of movement, and the manifestations of this condition require to be brought out either by the examination of the physician, or by some fortuitous circumstance. It is of the nature of paralysis in that it is generally concomitant with some muscular feebleness, and of the nature of contraction in that a very slight cause is sufficient to produce a contracture."

This state is common in hysteria; of 43 women and 27 men, M. Berbez (98) found 33 women and 19 men affected with it. It may be general or localized. M. Charcot observes that on the side on which a contraction develops there is almost always more or less anæsthesia, ovarian pain and some paresis, all of which have probably preceded the appearance of the contraction. Here we recognize an important law, that almost invariably disturbances of sensation of some kind exist in the domain of the contracted muscles, or are superimposed upon the contracture.

A simple means of provoking spasmodic muscular contraction is to tell the patient to squeeze the dynamometer with all his

force, the hand will then, in cases where the contraction diathesis is present, often remain contracted on the instrument.

The measures by which a contracture may be provoked in hysterical patients, are:—

(1) Excitation of the skin ; light stroking, tickling, superficial electrification, prolonged soft insufflation, &c.

(2) Excitation of the muscles: energetic voluntary contraction: pressure on muscular masses: repeated percussion of the muscles or their tendons; tetanisation by faradism; encircling the limb by a ligature: sudden flexion of a limb (paradoxical contraction).

(3) Irritation of bones or fibrous tissues; passive traction on joints, pressure or percussion on bones, &c.

(4) Psychological agencies corresponding to the different modes of suggestion in the waking state.

It is not necessary for the cutaneous excitations to be perceived for contracture to occur, they take effect on an anæsthetic limb. The contracture is not due to slight pressure exercised on the subjacent muscles, for it takes place when the excitation is made over the bone only, *e.g.*, the internal surface of the tibia. The spasm or contracture can be brought to an end by blowing upon the affected limb. When the spasm is generalised, or extends to a large number of muscular groups, it can be stopped by suggestion in hypnotizable subjects, or by provoking a convulsive seizure, either by means of pressure over the ovary, or by exciting the skin over the epigastrium. M. Pitres thinks that the fact that general muscular spasm can be induced by continued irritation of one spot shows that the stimulus acts through the ordinary reflex channels by the intermediary of the spinal gray matter. The persistence of the spasm, after removal of the stimulus, shows that it is not an ordinary reflex action, however, but rather is to be considered as the result of a lasting (durable) change of state produced in the gray matter by the action of the stimulus. Spasm may also be excited by applying (not too tightly) an Esmarch's bandage round a limb, *e.g.*, the forearm, for some time. After a little while, especially if the muscles are put into action by squeezing the dynamometer, spasmodic contracture of the muscles appears, putting the hand into the position of flexion from the predominating action of the flexors. If the bandage is applied too tightly the contraction ceases. When it is removed, as a rule the spasm disappears; if it persists, massage of the muscles will remove it. It may, however, become generalised. In this case, it may be limited to the muscles of the superior extremity only, or may spread to those of the

corresponding side of the face, trunk, and lower limbs: in this case there is generally anæsthesia of this side. Sometimes contractions may invade all four limbs and the whole face. When contractions have appeared spontaneously, or have been provoked on several occasions, they more readily become general. When a contraction, whether purposely excited or not, has lasted some time, massage often fails completely to remove it, whence the axiom of Charcot, "*qu'il ne faut pas laisser traîner les contractures.*"

M. P. Richer has made the following observations on the electrical reactions of the muscles in the contraction-diathesis.

(1) In faradisation with rapid interruptions, the muscles stimulated enter into tetanus and other (neighbouring) muscles contract in addition to those directly excited. (2) If the interruptions are slow (3 per second) they give rise to contractions which, at first distinct from one another, are rapidly fused into a state of tonic contraction or tetanus. To single shocks (1) the line of descent (in graphic records) at first rapid, becomes slow, and then descends more rapidly to the base line. (2) Permanent contraction may be produced by a single stimulation; the line of descent is cut short, and the muscle remains shortened. (3) As a rule several shocks are necessary to develop a permanent contraction. After a few shocks the contraction begins to develop, then each new stimulation augments the contraction, which now does not remain limited to the muscle directly stimulated, but invades all the muscles of the limb. MM. Brissaud and Richet have shown (99) that a muscle already in a state of spontaneous or induced contraction will contract still further on electrical stimulation. It follows that in this state of contracture there is not the maximum shortening of the muscle, but an intermediate state between tetanus and relaxation; this myographic tracings demonstrate. M. Pitres has applied this fact in clinical work. "Remark," says he, "that the right arm of our patient has remained in the position in which it was placed before being invaded by the contracture. The fingers and the elbow which were semiflexed at the beginning of the experiment, remain so; the wrist which was in extension, is still extended. This proves that the contracture is not necessarily accompanied by any considerable or marked shortening of the muscular fibres."

This state of readiness to contraction is associated with muscular weakness. In some cases the weakness precedes the appearance of the contraction, in others it follows it. That

the muscular feebleness is not caused by the contraction-diathesis is proved by the fact, that the weakness is often seen to gradually improve, whilst the "diathèse de contracture" persists in power and in activity long after the limb has recovered its normal strength.

Together with the contraction-diathesis another phenomenon ought to be classed amongst the permanent stigmata of hysteria. This is a state of muscular feebleness or "amyosthenia;" it is often the forerunner of paralysis. It may be limited to a part of a limb, *e.g.*, the hand, may invade the whole limb or affect, which is common, a hemiplegic type. Sometimes all four limbs are affected. Whatever the seat or extent of amyosthenia it never involves one muscular group to the exclusion of its antagonists. No "active" deformities are thus produced by it. But in the case of the lower extremity, the foot may drop by its own weight, or that of the superincumbent bed-clothes, and from after a time become fixed in this position by adhesions in the joints and tendons. Care must, therefore, be taken to prevent this. As in the case of the contracture-diathesis, disturbances of general sensibility may also be present in the domain of the muscles invaded by paresis. "The tendon reflexes of an amyosthenic limb are generally exalted, and there often co-exists this state of special aptitude to contraction" (Richer). M. Gilles de la Tourette, however, states that the paresis which accompanies the "diathesis of contracture" is independent of the "diathesis of amyosthenia" which is a clearly differentiated phenomenon, and that the reflexes in the latter are not, as a rule, exaggerated, but diminished. M. P. Richer says "that amyosthenia occurs most often on the left side; and that experiment has shown him that æsthesiogenic agents, which act on the sensibility, also affect amyosthenia, which is displaced, together with the anæsthesia which accompanies it, from one side of the body to the other, in the phenomenon of transference and its oscillations." This only applies when, as is the rule, sensory troubles are present.

Rhythmical hysterical spasms may be classified according to their seat as follows:—

(1) Rhythmic localized movements, limited to one muscle or group of muscles, and producing simple movements of flexion, extension or rotation of the head or limbs.

(2) Spasmodic co-ordinated movements of more complex character than the above, affecting several muscular groups synergically associated; hysterical chorea.

(3) Rhythmic respiratory spasms affecting the muscles of phonation and respiration.

The respiratory spasms may be divided into :—

(1) Simple, (*a*) inspiratory, (*b*) expiratory, the spasmodic movement occurring only during inspiration and expiration respectively.

(2) Mixed, in which both inspiration and expiration are modified by the spasm.

(3) Complicated, in which the muscles of respiration and phonation are both involved.

The rhythmical hysterical spasms must be distinguished from the “*maladie des tics convulsifs*” described by Charcot (100), Gilles de la Tourette (101), Guinon (102). This affection is hereditary, very obstinate, and characterised by the association of “tics” with diverse nervous affections such as coprolalia, the sudden involuntary utterance of filthy words; echolalia, automatic repetition of sounds or noises; echokinesis, repeated imitation of movements; fixed ideas; “*déire du doute*.” Hysteria may sometimes simulate it but the diagnosis may always be made by studying the antecedent history of the patients, the conditions under which the illness developed, and the symptoms with which it is associated.

General characters of rhythmical hysterical spasms.

(1) Etiology. They occur generally in children or young adults, hereditarily predisposed to hysteria or already affected with it. They nearly always follow strong moral emotions, injuries, or an hysterical convulsion. The onset is sudden and the primary character maintained throughout.

(2) The spasms affect one or several muscles; sometimes groups of muscles habitually associated in their action, sometimes the muscles of one side of the body are involved. They may be quickly or slowly repeated, always at a regular interval. Generally sudden, like the muscular contraction following an electric shock, they may sometimes be slow and sustained. They may be constant or occur in series. They cease during sleep, and may be suspended by certain measures, compression of certain parts of the body, drawing off of the attention, reading aloud or singing, &c. They are not accompanied by mental disturbance, nor by loss of consciousness. The patients are perfectly aware of the movements but cannot stop them.

(3) The distinction of a rhythmical spasmodic affection of hysterical origin from one not hysterical is to be made by determining the presence of hysteria on other grounds; such as the

exciting cause of the spasm, history of former hysterical affections in the patient or in his family, and the presence of the sensory stigmata of the neurosis.

(4) The course and duration of these spasms is very variable. If the patient is in a situation where she is brought into contact with other persons who are predisposed to become hysterical, *e.g.*, in a prison or a convent, she should be isolated, or otherwise one case may lead to an epidemic outbreak amongst those who witness it.

In describing *hysterical mutism and stammering* Prof. Pitres gives the case of a young man who stammered from the time that he saw his father killed under his eyes, in an accident. This patient presented concentric contraction of the visual fields, and large areas of partial anæsthesia over the legs and the upper part of the trunk. M. Pitres thinks that many cases of ordinary stammering are really of hysterical nature, judging from the conditions under which the affection develops, namely, sudden fright, shock, or injury, and the suddenness with which cure often takes place.

Hysterical Paralysis.—The following characters distinguish hysterical paralysis.

(1) It is a flaccid paralysis with preservation of tendon-reflexes and of contraction of the muscles on mechanical and electrical stimulation.

(2) It is not transformed into spastic or atrophic paralysis. The muscles may waste after a time, but never to the extent observed in muscular atrophies of spinal origin, and the wasting present corresponds to the disuse of the muscles.

(3) It is accompanied by defects of common sensation and of the muscular sense.

(4) It often disappears during hypnotic sleep and reappears on waking.

(5) It can be relieved or displaced by agencies which have no effect on paralysis due to organic disease, *e.g.*, convulsive attacks, suggestion, application of "æsthesiogenic" agents.

Hysterical hemiplegia. Varieties:

(1) Partial hemiplegia, in which the arm and leg on one side of the body are affected, but the face escapes.

(2) Total hemiplegia, in which certain motor affections of the face co-exist with paralysis of the limbs on the same side.

(3) Hemiplegia with paraplegia, in which one arm and both legs are affected with or without participation of the face and tongue.

(4) Alternate hemiplegia, paralysis of the limbs on one side, and of the opposite side of the face.

(5) Double hemiplegia, in which all four limbs are paralysed.

(i.) The first variety has been well described by Todd. "The face and the tongue escape; the paralysis, limited to the arm and leg, is often incomplete; the muscles are always flaccid and are not wasted; sometimes one or other of the paralysed muscles is the seat of muscular spasms or of cataleptic rigidity. In walking, if the paralysis is well-marked, the leg is dragged like a foreign body, and scrapes the ground."

(ii.) The total hemiplegias show a remarkable peculiarity, the face and the tongue, instead of being paralysed are the seat of spasmodic contraction, known from the researches of Charcot as hysterical glosso-labial hemispasm. This symptom is, according to Charcot, characteristic of hysteria.

Hysterical glosso-labial hemispasm.—In repose the mouth is asymmetrical, the labial commissure is drawn upwards and outwards, and the naso-labial furrow deepened on the affected side. The tongue lies on the floor of the mouth, deviating towards the affected side, and often cannot be protruded. When the patients can protrude it, it is strongly deviated and curved ("en crochet"), its tip directed away from the side of the facial spasm and paralysed limbs. M. G. Lombroso (103) has reported some cases of hysterical hemiplegia with paralysis of the lower part of the face, as in organic hemiplegia, but these require confirmation, and at present this glosso-labial hemispasm must be regarded as one of the most important symptoms of hysterical hemiplegia.

MM. Brissaud and Marie describe in the following terms, after the teaching of M. Charcot, the differences between hemiplegia of hysterical and organic origin. "In patients suffering from a localised organic cerebral lesion, the paralysed leg, instead of being, as in the normal state, carried directly forwards by flexion of the leg and thigh, can only be brought in front of the other by a movement of abduction, in which it describes a semicircle of which the sound foot is the centre. During this movement, the paralysed limb remains extended. In hysteria, on the contrary the paralysed leg is no longer brought forwards, but is dragged behind the sound limb. The patient walks in something the same way as children go upstairs, always with the same leg first. Further, during walking, the paralysed limb is often a little flexed, and not extended as in the case of organic hemiplegia."

(iii.) In alternate hysterical hemiplegia the side of the face or tongue opposite to the paralysed arm and leg is affected with spasm (glosso-labial hemispasm).

The most common complications of hysterical hemiplegia are œdema, muscular atrophy, and persistent (permanent) contractions of the paralysed limbs..

Hysterical Paraplegia. Varieties.—(1) True paraplegia, complete loss of power in the legs in all positions; (2) “abasie,” in which the paralysis only appears when the patients are in the vertical position. (1) Hysterical paraplegia may be of the flaccid or spastic kind.

(2) Under the name of “abasie” is described a form of hysterical paraplegia, in which the patients when lying down are able to perform any movement with their legs, but when standing are powerless.

MM. Charcot and Richer (104) described, in 1883, this special form of pseudo-paralysis of the legs, and M. Blocq gave a more extended account of it, and showed that this affection is ordinarily closely connected with hysteria, in the course of which it forms an episode, generally a passing one (105). It most frequently occurs in boys of 8 to 14 years of age, but may be seen in adults and in the aged. It comes on either slowly or suddenly; supervening generally on a moral emotion or slight injury. It is characterised essentially by the loss of the co-ordinated muscular movements by means of which equilibrium is maintained during standing and walking. When the patients sit or lie they can move their legs normally, there is then no loss of muscular power nor of the muscle-sense; the knee-jerk is normal, there is no ankle-clonus. When they try to stand or walk, their legs bend under them; they are incapable of holding themselves upright, and of moving forwards (abasie paralytique), or in other cases, their legs are the seat of tremors (abasie trepidante), or execute inco-ordinated movements resembling those of ataxics (abasie choréiforme). There are several varieties. M. Charcot classifies the cases as above (106). The divisions might be multiplied, but this would needlessly complicate the study of the disease. The acts of walking, leaping, climbing, dancing, &c., are all complex acts which we have acquired by long training. Each of them implies the co-ordinated action of groups of muscles associated by education to act together for this end. Each of these actions can be performed independently of the others, and corresponds to a distinct muscular mechanism which can be separately deranged. Just as a man can walk without being able

to dance or swim, so a patient may lose the power of walking, and yet be able to jump with his feet together, or to go on his hands and knees. Very clear examples of this are afforded by different cases of abasie (107). The prognosis is generally good. "Abasie" has lasted in reported cases from some days to fifteen months. Recovery is the rule, but relapses are common. M. Blocq thinks that the functional disturbance which produces "abasie" may be either an inhibition of the controlling cortical centre which puts into action the spinal mechanism that presides over the act of walking, or an inhibition of this spinal centre itself.

Hysterical pseudo-tabes.—In pseudo-tabes the characteristic lesion of the posterior columns of the cord is absent, but the patients present more or less faithfully the classical motor and sensory symptoms of locomotor ataxy. These symptoms may be due to cerebellar tumour, to the lesions of disseminated sclerosis, to peripheral neuritis, or lastly may not depend upon any organic lesion of the spinal cord or nerves as in certain cases of hysteria and of neurasthenia.

Hysterical and neurasthenic pseudo-tabes present the same group of symptoms.

There are on record ten well authenticated cases of hysterical pseudo-tabes, of which six were men and four women. The onset is slow, the course progressive, and the duration varies from some days to several years. In some cases temporary cure was followed by a relapse.

Symptoms.—The most constant were, instability in standing and motor inco-ordination in walking, increased on closing the eyes. The gait often exactly resembled that of true tabes dorsalis. Lightning pains occurred in half the cases; girdle pains were almost as frequent. There were also spinal pains and tenderness to pressure over the vertebral spines. Paræsthesie were sometimes noted in the lower extremities. In some patients there was hyperæsthesia, in others anæsthesia of the soles. The sense of position of the limbs was preserved. The plantar reflexes and knee-jerks were present. There was no Argyll-Robertson phenomenon nor atrophy of the optic discs. In one or two patients there were slight bladder and rectal troubles. The patients often presented in addition to the above symptoms the sensory stigmata of hysteria or the characteristic symptoms of neurasthenia. Hysterical pseudo-tabes then resembles true tabes dorsalis in the disturbance of equilibration and of walking, in the presence of Romberg's sign and of troubles of sensation, but differs from it in

the preservation of the knee-jerks and plantar reflexes, and in the absence of optic atrophy and of the Argyll-Robertson phenomenon.

Hysterical Tremors.

Contrary to the opinion of Briquet, who considered that hysterical tremors were a "convulsion en petit," M. Gilles de la Tourette thinks that they form part of the common basis of hysteria formed by the union of the permanent stigmata. Like the sensory troubles these tremors show great tenacity, persisting during several months, and at times intensifying so much as to form "attacks of tremor." These paroxysms are only the exaltation of the stigmata, and if the contraction-diathesis and the sensory troubles are accepted as permanent stigmata, tremor must be equally so accepted.

Many cases of hysterical tremor had been published but remained isolated until M. Charcot (108) undertook a systematic account of the subject, and was followed by Prof. Pitres (109), M. Rendu, and lastly by M. Dutil (110), in whose inaugural thesis the fullest account of the subject is to be found.

Their frequency is less than that of the disturbances of sensation or the contraction-diathesis, but further observations on this point are needed. M. Charcot teaches that hysterical tremor especially occurs in men, and is often present in cases of traumatic hysteria; meanwhile it is frequently seen in women and is not rare in children. Tremor, whatever form it may take, generally appears suddenly under the influence of a traumatism, fright or moral shock of some kind, and very frequently after a convulsive attack. When it exists previously, the occurrence of an attack increases it. It may be general or partial in distribution, may affect a whole limb or part of it, or assume a paraplegic or hemiplegic form. The duration is very variable. Tremor may last for months or years without any interruption. It may be slight or so severe as to render walking difficult and the ordinary uses of the arm and hands almost impossible. Classical authors say that paralysis agitans and senile tremor sometimes supervene suddenly on a strong moral emotion: the question of hysteria in such cases requires fresh investigation in the light of recent observations.

The tremor has generally a regular rhythm but the frequency of the oscillations is very variable, even in the same patient at different times. It may be remarkably slow, very rapid, or of medium rapidity, this latter being the most common form.

The following are the classifications adopted by M. Charcot and by M. Dutil; they do not differ essentially.

M. Charcot's classification.

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| A. Tremor, exaggerated by voluntary movements. | 1. Oscillating tremor with oscillations 3-6 per second. | Resembles paralysis agitans or senile tremor. |
| | 2. Oscillating tremor with rapid oscillations 8-9 or more per second. | |
| B. A tremor, which may or may not exist during rest, is provoked or exaggerated by volitional movements which do not accelerate it, but increase the amplitude of the vibrations. | 3. Intentional tremor (type Rendu) intermediate between 1 and 2 with regard to the frequency of the oscillations. | Resembles the tremor of disseminated sclerosis and still more perfectly mercurial tremor which according to M. Letulle is very often of hysterical nature. |
| | | |

M. Dutil's classification.

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| 1. Vibratory tremor 8-9 oscillations or more per second. | Persists during rest, is little or not at all modified by voluntary movement. | Resembles tremor of Graves' disease, of alcoholism, and of general paralysis. |
| | | |
| 2. Tremor of medium rhythm $5\frac{1}{2}$ - $7\frac{1}{2}$ oscillations per second. | a. Intentional remitting tremor (type Rendu). It may or may not exist during rest, and is exaggerated during voluntary movement. | Resembles exactly mercurial tremor and less perfectly that of disseminated sclerosis. |
| | b. A paraplegic form of the above. | |
| | c. Purely voluntary tremor, only appearing on voluntary movement. | |
| 3. Slow tremor 4-5½ oscillations per second. | Persists during rest, is little or not at all modified by voluntary movements. | Resembles paralysis agitans and senile-tremor. |
| | | |

The vibratory tremor No. 1 may be generalised or partial in distribution and as a rule lasts only for an hour or two being then only a post-convulsive phenomenon. In some patients, however, it may become chronic, and when well marked and general the patient is agitated by a constant trembling or vibration whether he is standing or sitting. When stripped the muscles of the trunk and limbs are seen to be agitated by little fibrillary

contractions, which in the fore-arms may determine small sudden movements of the fingers. When the tremor is at its maximum intensity as after a fit or some strong emotion the muscles of the face and lips share in the tremor, the speech is tremulous, and the aspect of the patient exactly recalls that of a person affected with general paralysis. In the quieter periods the tremor resembles that of Graves' disease. Periods of quiet alternate with periods of increase of tremor, which only entirely ceases during sleep. In the common run of cases, this tremor does not interfere with ordinary movements, except those which require a certain amount of precision such as writing, so that walking is ordinarily not interfered with, but during the exaggeration of the tremor which follows a convulsive attack a giving way of the legs occurs, and being frequently repeated, renders standing very difficult. Graphic tracings show that the oscillations are small, restricted in range, and of unequal amplitude.

The first variety of the tremors of middle range—intentional remitting tremor—resembles that of disseminated sclerosis but differs from the latter in being constant, meanwhile it may cease in the sitting or lying posture and during sleep. Is exaggerated during volitional movements. The patient's head, trunk and limbs are agitated by constantly repeated small rhythmical muscular contractions. If the patient is seated the head nods to and fro, the forearms and hands are agitated by slight movements of flexion with pronation followed by extension; whilst the feet tap against the floor with a noise resembling that made by an impatient person who taps with his toes. A voluntary movement of the limb exaggerates the tremor; if a glass of water is carried to the lips the water is spilt by the progressively increasing tremor; writing and similar fine exact movements are often rendered impossible. When the patient stands the oscillations become greater and more abrupt, and constant slight movements of flexion and extension of the legs at the knee occur. This tremor generally predominates in the limbs on one side of the body. It is increased by convulsive attacks, emotions, and irritation of hysterogenic zones. It may persist for years, or pass away more or less rapidly. Though further observations are necessary to form a conclusion, there is much evidence that mercurial tremor, which this variety closely resembles, is in the majority of cases of hysterical origin.

The second variety of the tremors of medium amplitude only differs from the foregoing in being confined to the lower extremities.

The third variety "purely voluntary tremor" exactly re-

sembles that of disseminated sclerosis. How is the diagnosis to be made between the latter disease and the hysterical manifestations which simulate it, and are accompanied by this peculiar tremor? The difficulty of diagnosis is often very great; no general rules can be given, each particular case must be considered on its merits. The presence of stigmata and a past history of hysteria, will be in favour of the neurosis, but it must be borne in mind that hysteria and disseminated sclerosis may be associated in the same subject, for M. Charcot has shown that there is no organic cerebro-spinal affection with which hysteria is more frequently combined than this one. Several cases have been published by Westphal, Langer, Babinski, and others, in which the diagnosis of disseminated sclerosis was made during life, but no lesion of any part of the nervous system was found at the autopsy. M. Gilles de la Tourette thinks that such cases should be placed under the category of hysteria. He further remarks "that this view has been recently combated by Dr. Buzzard. . . This author . . . recalls that a number of organic affections have been taken for hysteria . . . and insists on the diagnostic value of the *intentional* tremor which, he says, does not exist in hysteria. After the facts we have brought forward, we shall be permitted to say that this opinion is no longer tenable. And Dr. Buzzard concludes by saying that many symptoms attributed hitherto to hysteria belong to organic affections, and that the domain of the neurosis is destined to be limited, as the measure and extent of our knowledge of organic affections of the spinal cord increases" (111). On the other hand, M. Souques working at the subject from the opposite point of view (112), says: "We sustain here exactly the reverse thesis, and we allege that, in proportion as we become better acquainted with hysteria, so a number of unusual cases, referred to the organic diseases, will be found to belong to it. As for the cases where during life symptoms of hysteria have been noted, and referred to the neurosis, and where the autopsy has revealed organic lesions, we have obviously to do with those associations of hysteria with organic disease on the frequency of which M. Charcot has for several years insisted."

The third variety of tremor (of M. Dutil) is made up of slow oscillations, and almost exactly resembles the tremor of paralysis agitans. The general aspect and bearing of the patient may also strikingly simulate that of patients affected with the latter disease. These cases are, however, not very common.

All forms of hysterical tremor cannot be brought under the varieties of the foregoing classifications; some being variable

and changeful in character. They may be accompanied by choreic movements, and so simulate the tremor and choreiform movements which occur in certain cases of cerebral tumour. Lastly, there may occur in "normal hysteria" muscular contractions which may affect, separately or together, the trunk, head, or limbs. But these "rhythmical spasms" occur so often under the form of attacks (rhythmical chorea) that it will be better to consider them with the convulsive paroxysms of hysteria.

Trophic disturbances in Hysteria.—Œdema generally appears as a complication in contracted or paralysed limbs, but may rarely come on independently of any affection of motion. Sydenham pointed out the characters of the œdema of hysteria as being more marked in the morning than in the evening, as not pitting on pressure with the finger tip, and as often affecting only one limb. M. Pitres thinks that this exaggeration of œdema in the morning is not constant, and cannot be relied upon as a diagnostic distinction between hysterical œdema and that of cardiac or renal origin.

As a rule, hysterical paralyses are not accompanied by any alterations in the electrical reactions of the muscles. Sometimes, however, the paralysed muscles rapidly lose their excitability to both faradic and galvanic currents. It is even possible that, as in a case reported by MM. Gilles de la Tourette and Dutil, the reaction of degeneration may be present. Prof. Pitres has not seen an instance of this, but relates a case of hystero-traumatic paralysis of the right side of the face following a burn. The affected muscles showed marked diminution of excitability to both forms of current, but no reaction of degeneration; this was noted fifteen days after the onset of the paralysis, and did not stand in the way of a speedy cure. There was also spasmodic contraction of the elevators of the lower jaw, which almost completely prevented opening of the mouth. It has already been observed that in hysterical hemiplegia the facial muscles behave differently from the other muscles of the body, and it is curious that in this case of hystero-traumatic paralysis of the face some muscles were contracted, whilst others were paralysed. This association of paralysis and contracture may prove useful in diagnosis. Local destruction of tissues from trophic lesions are rare in hysteria; but the nails may spontaneously fall off, and the author relates a case which he thinks shows that the teeth may be lost, and a sacral bed sore form.

Nutrition in Hysteria.—Even till lately it was generally accepted as true, that although hysterical patients eat a very in-

sufficient quantity of food, and often suffer from digestive disturbances, their general state of nutrition remains good. M. Empereur (113) arrived at the conclusion that, although the quantity of food taken was insufficient, wasting did not occur because disassimilation was sluggish; the outgoings, the loss due to disassimilative tissue change, were small, and, therefore, in spite of the small quantity of food taken, the general nutrition was maintained.

MM. Gilles de la Tourette and Cathelineau have made observations on the state of nutrition in "normal" hysteria. They define the "normal" hysteric as a patient, who presents at the time of observation, no other manifestations of the neurosis than the permanent stigmata which mark him as the subject of confirmed hysteria. They selected, to put themselves above criticism, patients who had entered the hospital for hysterical convulsions, contractions, cough, mutism, &c., and were careful to make their experiments at a time when these symptoms could not influence the nutrition, *i.e.*, during the interval between widely separated attacks. The conditions under which the patients at the Salpêtrière live are, from the vast extent and general arrangements of the hospital, those of ordinary life. They found first that "normal" hysterical patients took a quantity of food quite sufficient to nourish perfectly a healthy person living under the same conditions. In their observations, 10 patients, 7 women and 3 men, were taken, their weight was ascertained, and the temperature taken every two days. The urine was collected from each patient for the twenty-four hours, measured, and at once analysed. The patients were directed to pass water before going to stool. During the period of observation, eight days in each case, the patient was placed under close surveillance. They found that in the hysteric, apart from manifestations of the neurosis other than the permanent stigmata, the nutritive processes are normally carried on, and the volume of the urine, and the urinary excreta bore the same proportion to the body weight as in the healthy.

From researches on the blood he concludes that—

(1) In a "normal" hysteric, the same solution of continuity of the integument as that made in a healthy person, gives issue to a quantity of blood about one-third less than the normal.

(2) Putting aside cases of anæmia and chlorosis, the number of the corpuscles, the quantity of hæmoglobin, urea, and glucose in the blood are normal.

The mental state of hysterical patients.—It is necessary, says

M. Charcot, to take hysteria as it exists, that is to say as a disease par excellence psychical. The ordinary mental state of hysterical patients must be considered, their manner of being, their mode of action, their reaction to their surroundings, their whole moral and intellectual behaviour. Most authors even up to quite recent times represent the hysterical woman as of an extraordinarily complex type, exceedingly versatile, remarkable for her spirit of duplicity, lying, and dissimulation. One common trait characterises them, said Tardieu, namely, instinctive simulation, an inveterate and constant necessity of lying without interest and without object, for the mere sake of lying. According to these writers, the psychologic state of hysteria includes almost all mental pathology, from the most evident acute delirium to the most characteristic chronic monomania. The labours of Charcot and of his pupils have done as much for the psychical as they have for the physical phenomena of hysteria. Nervous heredity is strong in the hysterical patient and if it is often homologous—mother hysterical, daughter hysterical—there is often also found in the family history evidence of insanity, epilepsy, &c. The conclusion follows that hysteria may, perhaps, more than any other disease, beyond its own proper mental state present the psychical stigmata of “degeneration” (dégénérescence).

When the errors of observation and false statements that have obscured the actual mental state in hysteria have been cleared away we find that the true condition becomes clear and may be expressed in one word, suggestibility, that is, extreme sensibility to suggestion. But if this condition of suggestibility is the one mental state it is not the less a complex one. The suggestions may come from outside, be extrinsic, or be intrinsic, the latter constituting auto-suggestion, a factor of extreme importance in hysteria.

What is the mechanism of this suggestibility? The convulsive attacks play a large part in it. The prodromal period must be included in these attacks; it may last for 1-3 days or longer, and throughout its duration the patients are restless and agitated. Some are excited and contentious, or laugh and cry without any reason, others are depressed, and seek solitude. Then come the convulsions, on the other hand the convulsion may abort, especially in children in whom, as M. Charcot has shown, it may take the form of mania and be composed of psychical prodromata with some contractions of the arms or twitchings of the eyeballs to alone represent the convulsive period.

These prodromata are not accurately speaking part of the

mental state of hysterics, they belong to the paroxysm, but in certain patients a succession of attacks may create a sort of permanent mental state. When these psychical manifestations appear, one tries to induce the threatening attack, and if the convulsive phenomena are not produced and the mental troubles persist for more than the usual time one can say that the attack will not come; it has had its psychical equivalent. In children this often occurs, but in the majority of cases the prodromata are accompanied by the delirium which characterises the fourth stage of the convulsive paroxysm. This delirium singularly influences the mental state in the intervals of the attacks. A profound impression—on account of the memories revived, for the delirium is most often a recalling of past acts of the life—is exercised on the brain by this delirium, and the hysterical patient is possessed for several days after the attack by this impression, which is generally a painful one. The hallucinations which end the attack are frequently so vivid as to give the patient the illusion that imaginary events have actually occurred. We have seen one of our patients, a man, after the end of the attack, get up suddenly, open the window and chase the purely visionary black cat that had tormented him. This delirium may directly produce physical troubles, as for instance, paraplegia in a patient who has believed himself to have fallen down a precipice. Numerous examples of this kind may be found in the history of epidemics of demoniac possession. On the other hand many phases of the delirium, which can be recognized by the attitudes and gestures of the patient at the time, are not remembered when she wakes; they have however modified the mental condition and the patient is in a state of joy or sadness, for which she can give no reason. Similar central processes explain the “*lacunæ of memory*” found in hysteria, through which a whole period of the past life may be forgotten one day to be recalled the next. Hallucinations of vision are particularly common, and have one characteristic which M. Charcot has brought out. The phantoms are not motionless but move in a direction which is always the same. The cats, rats, &c., run along passing before the patient from left to right, or from right to left, according as the hemianæsthesia affects the left or the right side. The hallucination always takes its departure from the anæsthetic side. Most often the phantom passes by the side of the patient; it comes from behind her to vanish in front of her on the *anæsthetic side* (114).

If auto-suggestion was only effected under the influence of attacks, the phenomena that it brings with it, and which occupy

so large a place in the mental state of hysteria, would be easy of interpretation. But we must take into consideration those cases of hysteria in which convulsive attacks are either absent or occur at long intervals. In this connection, the terrifying dreams and nightmares by which the sleep is disturbed in hysteria, and which Sydenham described, are of great importance in understanding aright the mental state. These nocturnal hallucinations are analogous to those of the attacks, but are much more frequent, occurring every night, whilst attacks are relatively rare. Further, the attacks can hardly pass unnoticed, whilst the hysteric is alone the witness of her dreams, and her friends do not know to what to attribute her changeful character, influenced as it is by the nature of the dreams that are often forgotten by morning. The instability of the hysterical temperament may be explained in this way, by its varying from day to day, according to the nature of the nocturnal hallucinations and their influence on the conduct of the following day. Paraplegia even may be determined by a prolonged dream, of which Féré has reported a remarkable instance (115). M. Pitres has also shown that the nocturnal hallucinations, like those of the attacks, may be accompanied by persistent painful sensations. "One of our patients receives, from time to time, an imaginary visit from an old woman, who touches her in different parts of the body, and on all the parts touched, hyperalgesic zones develop which last for several days."

The intensity of these dreams may even determine trophic troubles; and the author relates the case of a girl of 19, who, after being disturbed by terrifying dreams during the night, felt towards morning a sharp pain on the inner side of the right leg, and when dressing found there an oval patch of "redness:" there was also right hemianæsthesia especially marked in this leg. He attributes this subcutaneous hæmorrhage, in the absence of any evidence of a traumatism, or of a nocturnal fit, or of its existence on the previous evening—to the influence of a very vivid dream. A passage from the life of St. Theresa is quoted to show that she was similarly the subject of painful hallucinations.

The nocturnal dreams and hallucinations in the female, are sometimes of an erotic character; this is much less often the case in men, though they may be as much tormented by dreams as women. To the persistence of these hallucinations in the waking state is to be attributed all these stories of "incubi" and "succubi" which are so prominent in the epidemics of demoniacal possession in the middle ages. These erotic hallucinations are

generally accompanied by severe pains. From the medico-legal point of view, the importance of these erotic hallucinations is obvious, as innocent persons have been unjustly accused and condemned on evidence of this kind.

Auto-suggestions, similar to the preceding, which are derived from the hallucinations of attacks or of dreams, may arise during the waking state. In this case, they are associated very often with nocturnal hallucinations. The extreme impressionability or suggestibility of hysterical patients has been noticed by most authors, and some very striking examples of it are given by M. Charcot (116). The hysterical also show an extraordinary credulity, and in a general way resemble children. The troubles of sensation, the stigmata, have an influence on the general bearing of hysterics. If, for instance, a patient likes to eat raw onions and lemons, it may be because ordinary food does not affect her anæsthetic palate; nor does the conduct of the man, who walked in winter in the courts of the hospital, clad only in a shirt and waistcoat, seem so eccentric when it is remembered that he suffered from total anæsthesia, and that cold did not affect him.

With regard to the question of simulation in hysteria. The true malingerer is a scheming, active person; the hysteric who malingers is not conscious of simulation. She is a passive being, a photographic-plate which has registered impressions, and keeps them as she has received them, sometimes indeed amplified, but always unconsciously and in good faith. Certainly malingerers exist, and may be hysterical, but they do not simulate from the fact of their being hysterical. The hysterical brain does not lend itself to enduring combinations; it is the slave of the suggestion of the moment, the mould on which an unconscious suggestion forms, and if the hysteric sins against the laws of society, it is very often beyond her own proper act. M. Laurent has well said (117): "How does the hysteric become a criminal? Rarely of his own will. The initiation of the crime by no means generally comes from him, almost always a more powerful will has prevailed over his own. He is *par excellence* the being who responds most readily to impressions from without, and obeys them without restraint." Noteworthy is the indifference that some hysterical patients, who are suffering from a local manifestation of the neurosis, paraplegia for example, show to it. Once affected, they seem not to concern themselves about it any longer, and under these conditions it does not fail to persist. In such cases, in order to effect a cure, it is necessary that "one suggestion should oust the other." M. Charcot has

shown the nature of this process of cure, and of the mode of action of isolation (118); he has also pointed out that, even in the most marvellous cures the curative suggestion operates generally only incompletely, and not immediately. A paralysed arm, for instance, does not suddenly regain its normal muscular power, even when trophic disturbances are absent, and the sensory stigmata persist a longer time still after the motor troubles have disappeared.

Hysteria may be associated with mental degeneration, but hysterical insanity has no real existence. Beyond the peculiar mental state that we have described, with its sudden suggestions due to extrinsic suggestions or to auto-suggestions, under various forms of hallucination, there exist only states of delirium or prolonged "somnambulistic" periods, which in no way resemble chronic insanity. So we may, *a priori*, conclude that the hysterical patients in asylums have been placed there for acts arising from their mental degeneration, and not from the hysteria associated with it. Numerous examples sustain this view (119).

In children, hysteria takes, in the majority of cases, a transitory character; the patients are extremely impressionable, the affective element predominates. The brain is, in children, in the period of rapid evolution; impressions received are strongly felt, but do not leave, as a rule, persistent traces.

In male hysteria, the mental state in those who have become affected by the neurosis in infancy or adolescence, is one in which a morbid "suggestibility" is dominant. The general bearing may be said to be effeminate. They are romantic; often boasting themselves of sexual powers which are imaginary.

Another type is more common in these cases, in which the psychical state of neurasthenia is added to that of hysteria. Intellectual impairment, cerebral apathy co-exists with lapses of memory, which render all work impossible. These lapses of memory are remarkable enough to lead to a suspicion of lying or malingering.

This mental state of hysterio-neurasthenia is not peculiar to men, but is found in women, in whom the manifestations of hysteria first appear at the menopause. In old age, the hallucinations, which formerly existed and bore witness to an excessive cerebral activity disappear, and the mental condition is one of sadness and melancholy.

On suicidal attempts in Hysteria.—Many authors have considered the suicidal attempts made by hysterical patients as not serious; they think that the latter have no intention of actually

carrying them out, but make their preparations with the view of attracting sympathy from, or evoking astonishment in, their neighbours (120). They hold that, generally speaking, the suicide of hysterics is a comedy, an act of simulation skilfully considered and prepared. M. Pitres, however, analysing their own observations shows that even these are sometimes in contradiction with this theory of simulation, as several of the attempts at suicide terminated in death. He then gives an account of eleven attempts at suicide in his 100 patients, and concludes that in hysteria suicide is generally the result of a sudden determination, taken without reflection, but that there is no sufficient reason to consider it as a comedy, played by the patients to make themselves interesting and to alarm their friends and neighbours. If the attempts at suicide made by hysterics do not often end fatally, the reason is that they are commonly not premeditated. The melancholics who desire to die, select a long time in advance, the moment and the means favourable for the execution of their project. The hysteric does not reflect. For a mere contrariety or trifling disappointment, she takes the resolution of killing herself, and thereupon swallows any poison that is at hand, or throws herself into the water. Next day, she is delighted at having escaped death. Facts of this nature are to be explained by the puerility of the hysterical character, passing as hysterical patients do like children, in one moment from grave to gay, from gentleness to violence, or from love to hate. With regard to the question of simulation, Prof. Pitres thinks that it is much less frequent than has been and is now stated. Hysterical patients relate what they feel without regard to the consequences. The unbridled love of deception which has been attributed to hysteria is a legend without serious foundation, as common sense and clinical observations show. It is impossible to suppose that the so-called witches who went to the stake for having stated that they had ridden to a "Sabbath" on a broomstick, or that, as has happened in our days, a patient, who had an hysterical club-foot amputated, could have been lying, in the first case, merely to gain notoriety, in the second, to enjoy the credulity of the surgeon.

General treatment of Hysteria.—Prophylactic treatment is very important; when a child shows the first signs of a neuropathic constitution, the symptoms of which in childhood have been already mentioned, the morbid tendency should be corrected by judicious physical and moral education. All forms of exercise which develop the muscles and strengthen the body are useful. Intellectual over-work, violent emotions, *e.g.*, terrifying stories of

ghosts, or giants, in short, everything which excites their sensibility and imagination is harmful. Children predisposed to hysteria are far better brought up away from their parents at a good school.

Before undertaking any plan of treatment the physician should fully gain the confidence of his patient, he should assure him that the malady from which he is suffering is not a grave one. That his symptoms are not uncommon, that they will pass away after a short time even without treatment, but that they will disappear more quickly on the carrying out of proper therapeutic measures. When the patient is surrounded by affectionate and at the same time injudicious friends, who are terrified at the least incidents of the malady and confirm him in his belief that he is suffering from an extraordinary and incurable disease (parents are often the most injudicious in this respect), then *isolation* from the friends is of the first importance. When the patient is isolated he will be placed in the most favourable position for deriving advantage from further treatment which should be directed (1) to bring to an end as speedily as possible the actual symptoms of the neurosis present and (2) to combat the morbid susceptibility from which these symptoms arise.

The measures to be employed for the second object are:—

Hydrotherapy, which is one of the most powerful agents in nervous affections. It may preferably be employed in the form of a cold douche, over all the body for a duration of 20-40 seconds, followed by a jet of hot water over the feet, and energetic rubbing with a dry towel. If this is not borne warm baths at 34°C for 45-60 minutes, or colder baths (30-28-26°C) of 5-15 minutes duration may be employed.

Static electricity, recommended especially by Prof. Charcot, demands costly apparatus and has other disadvantages. Drugs are very rarely of any use. Iron, arsenic and quinine are useful when chlorosis is present but have no direct effect on the neurosis. Morphia is undoubtedly effectual but unless used with great caution the remedy may be worse than the disease.

As for hypnosis (121) paralyses, contractures, rhythmical spasms sometimes disappear with marvellous rapidity under the influence of suggestion, but it does not cure the hysteria itself. All hysterical patients cannot be hypnotised, and of those who can be hypnotised all are not amenable to suggestion. Further this method is not always harmless; certain subjects become, after some séances of hypnosis, so sensible to hypnotisation that a

look, gesture, or unexpected noise is sufficient to throw them into a state of hypnotic sleep. In short, hypnosis is a mode of treatment difficult to manage and which should be reserved for special cases. If a patient has been previously hypnotised by others or is subject to attacks of (hypnotic) sleep, hypnotic suggestion may be tried. The author does not think it well to go further, nor to commence treatment by hypnosis unless at the most pressing request of the patients or of their friends.

None of the operations on the generative organs which have been proposed and carried out are justifiable.

If asked as to the question of marriage as a cure for hysteria it is well not to take the responsibility of advising it.

Additional Note on Nutrition in Hysteria.

We may add here that the statements of MM. Gilles de la Tourette and Cathelineau, which have been quoted above, as to the changes in the urinary excreta after paroxysmal manifestations, have been criticised by other observers, and the question of the value of these changes in differential diagnosis must, perhaps, at present be regarded as *sub judice*. M. Voulgres, in a thesis (122) on this subject, finds that intellectual work and fatigue increase the secretion of the earthy, muscular work that of the alkaline phosphates, so that in a healthy man fatigued by a series of efforts, there is a tendency to inversion of the normal ratio. He agrees with the above authors that the proportion of the earthy to the alkaline phosphates tends to become, 1-2 equal, or even 2-1 during the twenty-four hours following an hysterical convulsive seizure, but says also that there is a similar, but less marked tendency, in non-convulsive hysteria, the proportion being 3 to 1.5. He further states that this inversion of the normal formula is not a pathognomonic sign of either convulsive or non-convulsive hysteria, for he has found it in other diseases, in paraplegia epilepsy, neurasthenia, locomotor ataxy, and phosphatic diabetes without nervous symptoms. He concludes that inversion of the ratio of the earthy to the alkaline phosphates is the *rule* after an hysterical, the *exception* after an epileptic fit.

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